APRIL, 1951

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Gastrointestinal Function following Radical Pancreaticoduodenectomy

The Choice of Treatment in Idiopathic Dilatation of the Esophagus

Clinical Manifestations and Surgical Treatment of Congenital Types of Diaphragmatic Hernia

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Sixteenth Annual Convention
Chicago, Ill., 17, 18, 19 September 1951

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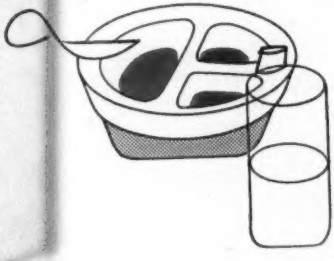

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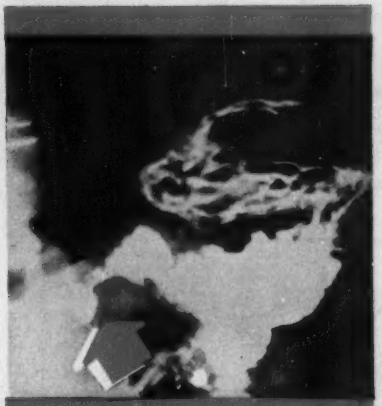

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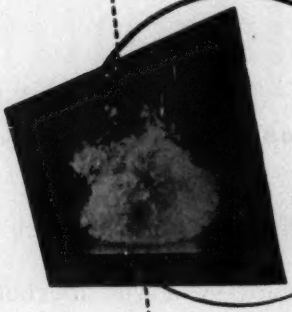

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The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

VOLUME 18 APRIL, 1951 NUMBER 4

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of the

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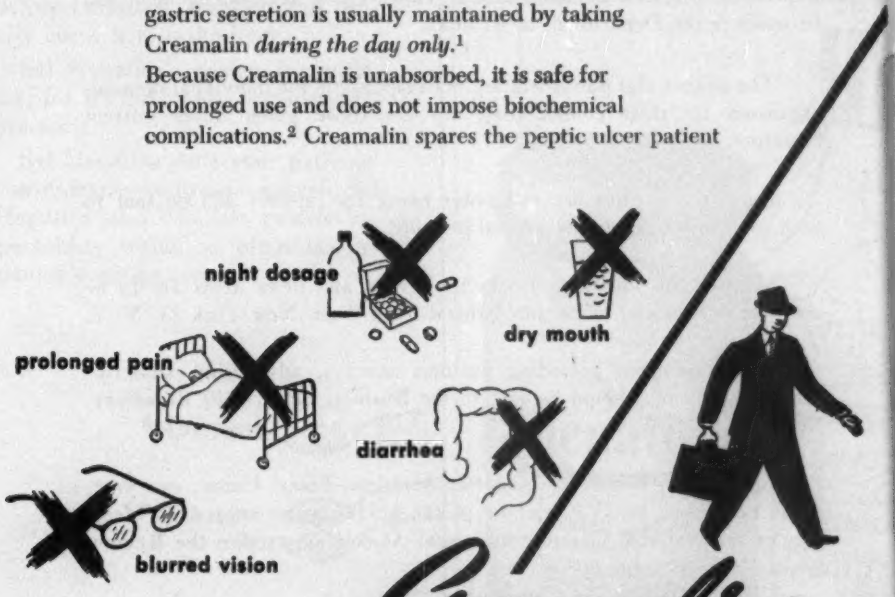

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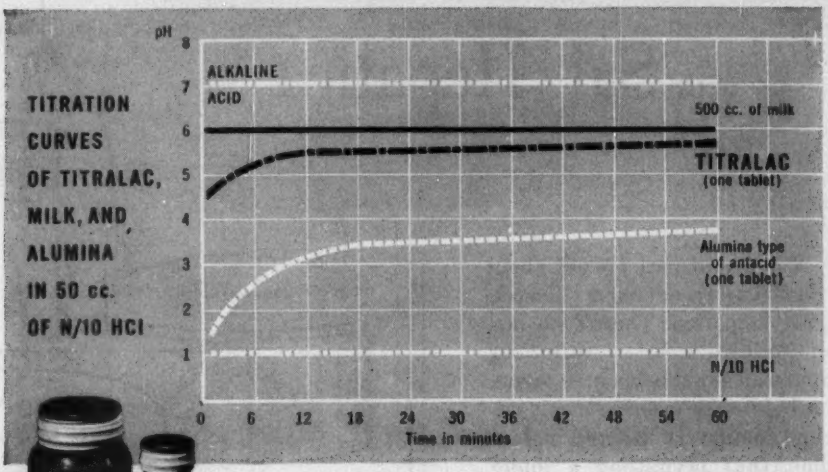

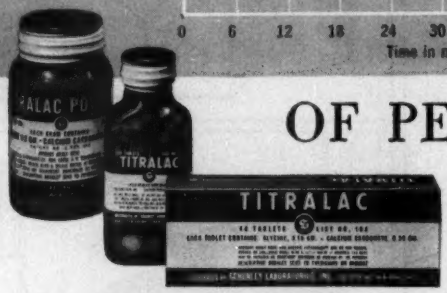

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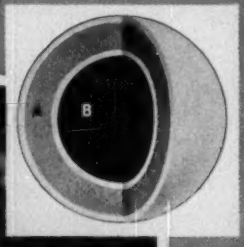

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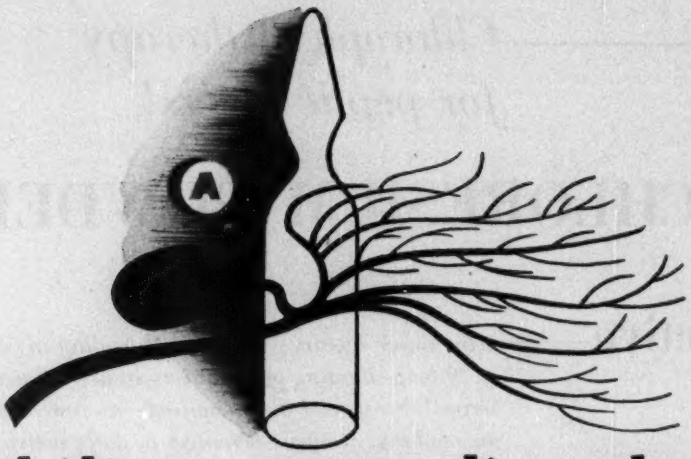

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Volume 18 APRIL, 1951 Number 4

CLINICAL MANIFESTATIONS AND SURGICAL TREATMENT OF CONGENITAL TYPES OF DIAPHRAGMATIC HERNIA*

STUART W. HARRINGTON, M.D.†
Rochester, Minn.

I am pleased to have the privilege of discussing with you the clinical and surgical considerations of some of the different types of diaphragmatic hernia, particularly those attributable to congenital structural deficiencies of the diaphragm and of the esophagus. The commoner herniae of this type are: (1) those which are due to a congenital absence of a portion of the diaphragm, (2) the pleuroperitoneal hiatus herniae (foramen of Bochdalek), (3) those due to congenital defect of an

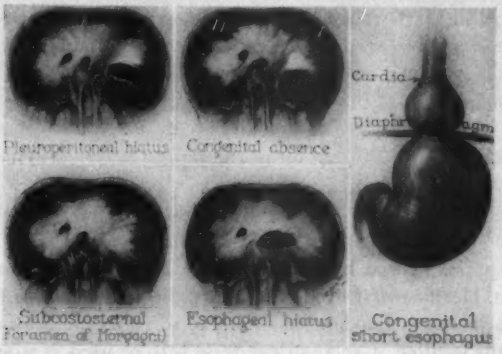

Fig. 1—Situations of embryonic defects of the diaphragm and esophagus which cause the more common types of congenital diaphragmatic hernia.

anterior portion of the diaphragm (foramen of Morgagni or subcostosternal), (4) some types of esophageal hiatus hernia, and (5) congenital short esophagus with partial thoracic stomach (Fig. 1). It is often difficult or impossible to determine definitely the type of hernia which is present on the basis of the clinical manifestations, but these findings are of great importance in determining the urgency and type of treatment to be instituted. The reason for the several different types of congenital diaphragmatic hernia due to structural deficiency of the diaphragm is the

*Read before the Fifteenth Annual Convention of the National Gastroenterological Association, New York, 9, 10, 11 October 1950.
†Division of Surgery, Mayo Clinic, Rochester, Minnesota.

unusual embryonic formation of the diaphragmatic muscle which makes it susceptible to weak areas through which these herniae may occur.

The formation of the diaphragm from embryonic structures is a highly complex process, because its muscular elements are derived from several embryonic structures. There is difference of opinion among embryologists as to the amount of diaphragm derived from each embryonic source.

The anterior, lateral and central parts, which comprise the greater portion of the diaphragm in the adult person, are formed from the transverse septum and fused ventral mesentery. The remaining posterolateral portion is formed by the fusion of the dorsal mesentery and the mesoderm derived from the receding wolffian body, and the pleuroperitoneal membrane derived from the pulmonary ridge. It is difficult to determine the exact amount of the muscle tissue that is derived from each of these structures, since considerable variation probably occurs during the process, but it is likely that the dorsal mesentery forms the posterior and central portions which contain the esophageal opening. The mesodermal cells from the receding wolffian body form the right and left crura. The pleuroperitoneal membrane grows ventrally, closes the remaining opening between the peritoneal celom and the pleural celom by fusion with the transverse septum and forms the posterolateral portion of the diaphragm.

Failure of fusion or failure of proper deposition of the mesoderm at any one of these adjacent points of union may result in congenital continuity of the pleural and peritoneal cavities or a congenitally weak portion in the diaphragm at any of these points. Consequently, from an embryologic standpoint, weak areas might be expected to appear at the points of fusion of these different structures. These areas are situated dorsolaterally at the pleuroperitoneal fissure (foramen of Bochdalek); also through the outer crus and through the esophageal hiatus. Unilateral absence of the diaphragm probably is the result of the failure of development of the pleuroperitoneal membrane, which usually is found as a narrow ridge of tissue along the posterior wall of the thorax. In the middle portion of the diaphragm anteriorly, on either side of the sternal attachment in the midline, there are two small apertures, called Larrey's spaces, caused by deficiencies in the structure of the musculature of the diaphragm. Herniae that occur through these spaces are usually called foramen of Morgagni herniae. Inasmuch as herniae of this type may occur anteriorly on either side of the midline, if an anatomic term is to be used, it would be preferable to designate them as "subcostosternal diaphragmatic herniae".

Herniation through the dome of the diaphragm is not common and cannot be explained on the foregoing basis, because the dome, embryologically, is not a fusion region. Such a hernia may be the result of excessive degeneration of the muscle in the formation of the central tendon or of some pathologic condition. Most herniae occurring through the esophageal hiatus can be attributed to an embryonic malformation of the muscle rim of the esophageal opening in the diaphragm, which in turn can be attributed to an abnormal formation of the esophagus in relation to the formation of the diaphragm. Congenital short esophagus with partial or complete thoracic stomach may be considered a type of esophageal

hiatus hernia. This condition results from the esophagus not developing sufficient length to reach the diaphragm, so that varying amounts of the stomach are suspended above the diaphragm. Ulceration is usually present at the esophagogastric junction and there often are varying degrees of stenosis.

Pleuroperitoneal Hiatus Herniae (Foramen of Bochdalek) and Congenital Absence of the Diaphragm

These two types of congenital diaphragmatic herniae are the most common types due to structural deficiencies of the diaphragm. They are essentially the same type and may be considered together, the difference being that of degree of malformation of the diaphragm.

The pleuroperitoneal hiatus herniae occur in the posterolateral portion of the diaphragm and are caused by failure of fusion of the pleuroperitoneal membrane

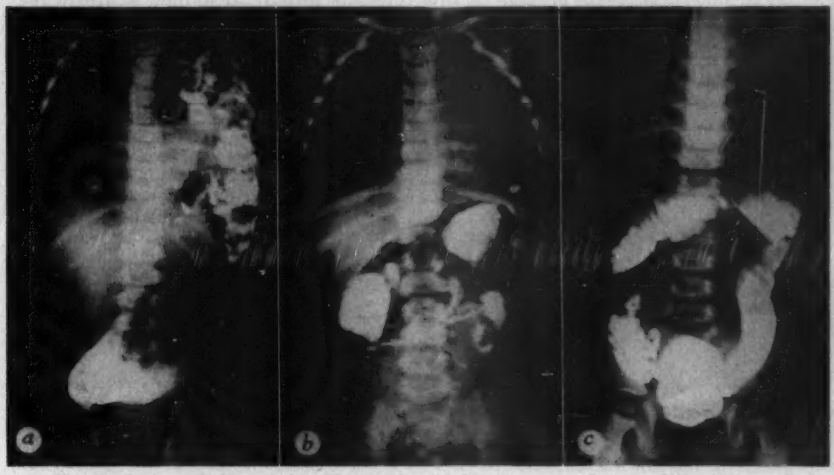

Fig. 2—Boy aged 11 months. Pleuroperitoneal hiatus types of congenital absence of the posterior portion of left diaphragm. Herniation of right colon, small bowel and spleen into left side of the thorax. *a*. On admission showing colon and small bowel filling most of left thoracic cavity. Markedly dilated stomach extends into pelvis. *b* and *c*. Eight months after abdominal repair of hernia. Entire stomach and colon in normal position in the abdomen. The diaphragm is normal in position and contour.

and the septum transversum. The defect is usually triangular with the apex toward the median portion of the diaphragm. The defect usually extends to the thoracic wall but occasionally there is an imperfectly developed band of muscular tissue extending along the thoracic wall. These herniae do not have a hernial sac and a direct communication exists between the abdominal and the thoracic cavity.

The commonest abdominal viscera involved in this type of hernia are the colon and the small bowel. There may or may not be herniation of the stomach and spleen. There are usually nonrotation of the colon and involvement in the hernia of the entire right portion of the colon (cecum and appendix), terminal ileum and small intestine to the jejunum. In many instances the stomach is not

involved in the hernia but it may be markedly dilated and extend into the pelvis (Fig. 2).

In the congenital absence of the posterior portion of the diaphragm, the hernial opening is much larger than that in the pleuroperitoneal hiatus type of hernia. This type of hernia results from failure of formation of that portion of the diaphragm which is derived from the pleuroperitoneal membrane. The defect is in the posterolateral portion of the diaphragm and usually extends from the eighth rib posteriorly and medially to the esophageal hiatus. These herniae usually do not have a sac but there may be an imperfectly developed enveloping membrane of peritoneum and omentum which simulates a sac. There are more abdominal viscera involved in the hernia than are usually found in the pleuropenitoneal hiatus type. These herniae usually contain stomach and spleen as well as portions of the large and small bowel. In some herniae of this type the ipsilateral kidney is above the level of the diaphragm in the thoracic cavity posterior to the pleura. I have noted the kidney in this position only when there has been complete absence of the diaphragm posteriorly. The presence of the kidney in the thoracic cavity can often be determined clinically by taking an intravenous pyelogram. This procedure is often helpful in distinguishing between a pleuroperitoneal hiatus hernia and complete absence of the diaphragm posteriorly. In many herniae of this type the left lobe of the liver is in the thoracic cavity and in some instances the pancreas is involved in the hernia.

Herniae of this type may occur in either the left or the right side of the diaphragm but are much more common in the left than in the right hemidiaphragm. The kind of abdominal viscera involved in the hernia depends on whether or not the defect occurs in the right or in the left hemidiaphragm and the severity of the subjective symptoms depends on the type of viscera involved.

These herniae are usually present at birth, at which time the respiratory and cardiac symptoms are usually most severe owing to the marked unilateral alteration of intrathoracic pressure and the occurrence of this derangement of intrathoracic pressure at a time when the compensatory cardiac and respiratory reserve has not been developed to a sufficient degree to maintain proper function of these organs. Many infants born with these congenital defects in the diaphragm die in the first few hours or days of life from what is thought to be cardiac or respiratory failure. If the respiratory and cardiac mechanisms are able to compensate for the presence of these abdominal viscera in the thorax, however, these patients may live on to childhood or even into adult life without any great amount of disability or many symptoms, providing intestinal or gastric obstruction does not develop. When the stomach is involved in these herniae and there is partial obstruction, the stomach becomes greatly dilated. These patients will give symptoms of partial gastric obstruction. Intestinal obstruction may occur because of bands of adhesions between the omentum and loops of bowel or because of inflammatory conditions of the bowel. In those cases in which there is a nonrotation of the right portion of the colon and the appendix is in the left side of the thoracic

cavity, appendicitis may develop and present a serious hazard to life. A ruptured appendix is usually associated with a fatal outcome. I have seen one such case.

Surgical Treatment:—These types of congenital herniae are present at birth. Inasmuch as many of the infants suffering from them die in the first few days of life because of respiratory and cardiac embarrassment, surgical treatment should be instituted immediately on the establishment of the diagnosis. In some instances these infants are able to survive in spite of the altered intrathoracic pressure and thoracic visceral relationships. In these cases, surgical intervention should be instituted as soon as possible because of the danger of intestinal obstruction. If the infants are able to maintain nourishment it may be advisable to delay surgical treatment for two or three months in order to permit some development of their accessory respiratory mechanism. If operation is delayed for a long period, how-

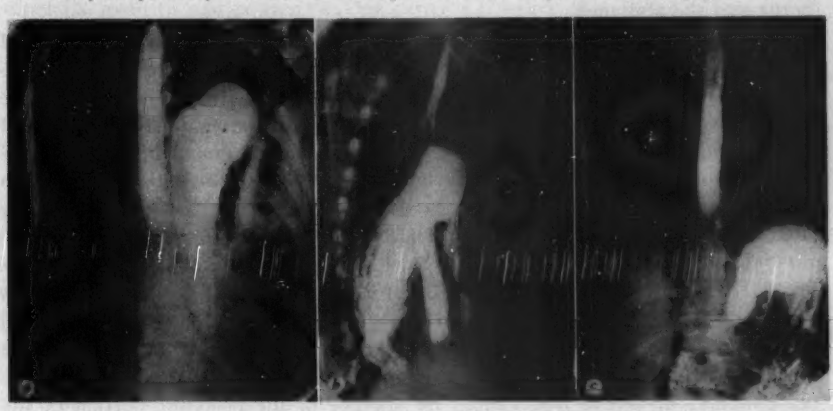

Fig. 3—Man aged 31 years. Congenital absence of posterior portion of diaphragm with herniation of the entire stomach, transverse colon, small intestine and spleen into the left side of the thorax. *a* and *b*. On admission of patient. Herniation of entire stomach into posterior left portion of the thorax and of colon into anterior portion of thorax. *c*. Four months after abdominal repair of hernia showing the entire stomach and colon below the diaphragm in normal position and contour. (From Harrington, S. W.: Diaphragmatic Hernia. In Nelson's Monographs on Surgery, 1950. In press.)

ever, the abdominal viscera will have lost their right of residence in the abdomen in that the abdominal cavity will not have developed sufficiently to contain them and there will be a marked increase in intraabdominal pressure when the viscera are replaced in the abdomen (Fig. 3). I have operated on several patients who have attained adult life with this type of hernia. The technical difficulties are greatly increased because with the lack of right of residence of the abdominal viscera in the abdomen, when the viscera are replaced, there is markedly increased intraabdominal pressure which causes considerable abdominal distention and also fixation of the diaphragm as well as respiratory difficulty in the immediately postoperative course. In these cases it is very important that the patient reduce weight so as to provide a place in the abdomen to contain the herniated viscera when they are replaced.

In the surgical treatment of these herniae the approach may be either thoracic or abdominal, but I prefer the abdominal approach through a left rectus incision.

In repair of the smaller herniae of this type, the opening can be closed without interruption of the phrenic nerve. On the other hand, in repair of the larger herniae interruption of the phrenic nerve is a necessary procedure. The opening is completely closed by overlapping the margins from 2 to 3 cm. If the patient is an infant, this closure is made with interrupted silk sutures. If the defect is too large to permit the relaxed diaphragm to span this gap, it is necessary to shorten the diameter of the diaphragm by extrapleural rib resection. Before the opening is completely closed, the air is aspirated from the pleural cavity by inserting a catheter connected to a suction apparatus. At the time of withdrawal of the catheter the last suture is tied, completely closing the communication between the thorax and the abdomen.

One of the chief dangers associated with the repair of these herniae is marked alteration of intrathoracic or intraabdominal pressure. It is very important in these cases that the respiratory function be maintained by positive pressure during the operation and that at the completion of the operation, negative pressure be secured and maintained in the thoracic cavity. A roentgenogram should be taken at the completion of the operation to see that there is no shift of the mediastinum due to the pneumothorax. I do not permit the patient to be moved from the operating table until I have seen the roentgenogram. If there is any shift of the mediastinum, more air is withdrawn to maintain the mediastinum in the midline.

Surgical Results:—In a series of 534 of various types of diaphragmatic hernia which I have operated on, 25 were due to structural deficiencies in the posterolateral portion of the diaphragm. Twenty-four patients had herniae due to complete congenital deficiency of the diaphragm, either of the pleuroperitoneal hiatus type (11 patients) or of the type caused by absence of the posterior portion of the diaphragm (13 patients). The herniae of these 24 patients were not contained in a hernial sac and the herniated abdominal viscera were in direct contact with the thoracic viscera. One patient had a hernia through a structural deficiency of the muscle of the diaphragm (foramen of Bochdalek) in which the peritoneum and pleura had covered the opening. There was a hernial sac, and the patient was 50 years of age.

The ages of the 24 patients with complete congenital deficiencies varied from 5 weeks to 33 years. Of the 25 patients 18 were male and 7 were female. In 24 cases, the herniae were through the left half of the diaphragm and 1 was through the right half. The abdominal approach was used for 23 patients and the thoracic approach for 2.

The structures involved in the hernia varied with the size and type of structural defect. The commonest structures involved were the small bowel, which was involved in 23 cases, and the colon, which was involved in 22 cases. The cecum and appendix were involved in 9 cases, the stomach in 11 cases, the spleen in 13 cases, a kidney in 7 cases, the liver in 3 cases and the pancreas in 1 case. In 2 of the 3 cases in which the liver was involved, the left lobe of the liver had herniated with

other abdominal viscera and in 1 case the hernia was on the right side and there was herniation of a portion of the liver only.

Nineteen patients underwent interruption of the phrenic nerve in conjunction with repair of the hernia. The spleen was removed from 3 patients and the appendix from 2 because of acute appendicitis. Thoracoplasty was done on 1 patient to narrow the diameter of the thorax and to facilitate the closure of a large defect in the diaphragm.

In my experience, the surgical repair of this type of hernia is associated with the highest mortality rate of any type of diaphragmatic hernia, for in 534 diaphragmatic herniae treated surgically there were 17 deaths, of which 7 were in these types of hernia, particularly those with large complete structural defects in infants. Two of these deaths were due to acute appendicitis with rupture of the appendix, pleuritis and peritonitis. One patient, 27 years of age, died of mesenteric thrombosis. One patient, an infant, died on the operating table before the opening in the diaphragm had been exposed. Three died from respiratory and cardiac failure, one of whom had a recurring hernia. In the 18 patients who survived the operation there has been no recurrence of the hernia and the patients have had no return of symptoms.

Herniae Through the Foramen of Morgagni (Subcostosternal)

These herniae are essentially direct herniae through a congenital defect in the structure of the diaphragm or a faulty attachment of the diaphragm to the sternum and costal cartilages. They are usually unilateral with the opening close to the attachments of the right costal cartilages to the sternum, but they may be bilateral with the small opening on the left side.

There is some difference of opinion as to whether these herniae should be classified as congenital or acquired herniae. It is impossible to explain their occurrence on a basis of faulty fusion or improper disposition of the embryonic mesodermic elements which go to form the diaphragm, as this anterior portion of the diaphragm is derived from the septum transversum only. But the consistency of the location of the hernial opening, the fairly constant relationship of the neck of the hernial sac to the round and falciform ligaments of the lever and the frequency with which the hernial sac protrudes into the right side of the thoracic cavity at the same point, that is, at the cardiophrenic angle, as well as the often associated nonrotation of the right portion of the colon, all strongly suggest a fundamental embryologic basis for these herniae. The constant presence of a peritoneal sac shows that the peritoneum had closed off the abdominal cavity from the pleural cavity before the actual herniation of the abdominal viscera occurred. The hernial sac of peritoneum is at the right cardiophrenic angle anteriorly, and as the hernia increases in size, the sac extends into the right side of the thoracic cavity.

Subcostosternal diaphragmatic hernia, in my experience, is one of the two types of diaphragmatic hernia which have a hernial sac. The other type of diaphragmatic hernia which has a hernial sac is that through the esophageal hiatus. It is interesting that subcostosternal hernia is probably the rarest type of diaphragmatic hernia and esophageal hiatus diaphragmatic hernia is the commonest; both are

essentially congenital in origin but are rarely present at birth, and occur in most instances in later life because of increased abdominal pressure on a congenitally defective diaphragm. I have operated on 3 patients who have had both subcostosternal and esophageal hiatus herniae. In 2 of these cases the subcostosternal hernia was bilateral.

The abdominal viscera usually involved in subcostosternal hernia are the colon, omentum, ileocecal coil and rarely the stomach.

Symptoms:—The subjective symptoms associated with these herniae are often indefinite and depend on the kind and amount of abdominal viscera involved in the hernia. There are essentially two types of subcostosternal herniae: (1) those in which the omentum is the only abdominal structure involved in the hernia, and (2) those in which the colon or the stomach is involved in the hernia. In the cases

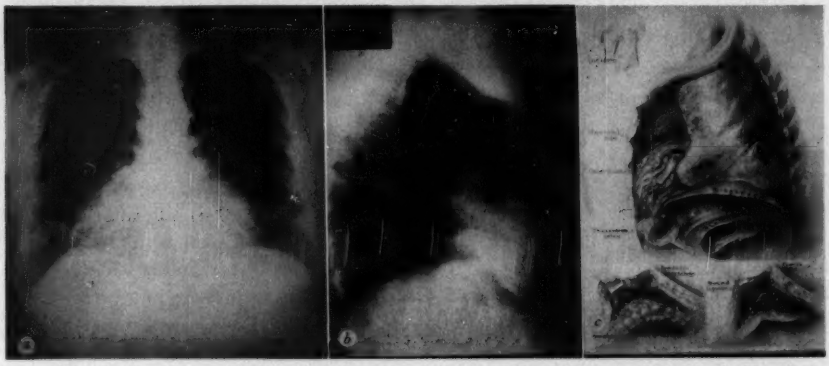

Fig. 4—Woman aged 35 years. Subcostosternal diaphragmatic hernia with herniation of the omentum into the right side of the thoracic cavity. *a* and *b*. On admission of patient, showing well-circumscribed lesion in right cardiophrenic angle extending into right anterior portion of thorax. Previously diagnosed intrathoracic tumor and enlarged heart. *c*. Herniation of the omentum through a defect in the anterior right portion of the diaphragm (foramen of Morgagni). The omentum is contained in a hernial sac of diaphragmatic peritoneum and pleura. Upper left insert shows abdominal approach for repair of hernia through upper right rectus incision. Lower left insert shows entire colon below diaphragm. Lower right insert shows repair of the defect in the anterior right portion of the diaphragm (foramen of Morgagni) with interrupted silk sutures and continuous fascia lata. Round ligament of the liver is sutured to the completed closure.

in which the omentum is the only structure involved in the hernia, the symptoms are those essentially related to the thoracic cavity, such as dyspnea, cough and pain in the lower part of the thorax. In those cases in which the hollow viscera are involved in the hernia, the symptoms are those of partial obstruction of the viscera involved as well as abdominal and thoracic pain.

The cases in which the omentum only is involved in the hernia present a much more difficult clinical problem in arriving at a definite diagnosis than those cases in which hollow viscera are involved in the hernia (Fig. 4). The subjective symptoms usually suggest a primary intrathoracic lesion because of the mechanical interference with respiration and expansion of the lungs caused by herniation of the omentum into the thoracic cavity. These symptoms direct clinical investigation to the thorax, and the roentgenographic thoracic findings of an area of increased

density in the pulmonary field justify the clinical diagnosis of a primary intrathoracic lesion, which may be thought to be an intrathoracic tumor. The possibility of this erroneous clinical diagnosis is of considerable importance in determining the type of surgical treatment to be instituted, because if the lesion is due to an omental diaphragmatic hernia, it is best to approach it through the abdomen, and obviously, if it is due to an intrathoracic tumor, it should be treated through a thoracic approach. This chief error in clinical diagnosis emphasizes the importance of careful consideration of all lesions in the right cardiophrenic angle.

Surgical Treatment:—The treatment of these herniae is surgical closure of the abnormal opening in the diaphragm after replacement of the abdominal viscera in the abdomen. I prefer the abdominal approach through the upper part of the right rectus muscle because the opening in the diaphragm is accessible and the abdominal contents of the hernia are more safely and easily reduced from the abdominal than from the thoracic side of the diaphragm, since the true relationship of the herniated viscera to the hernial sac can be accurately determined.

The method of closure of the neck of the sac and of the defect in the structure of the muscle of the diaphragm depends on the size and character of the opening. Small linear openings may be closed by overlapping the margins. Larger transverse openings extending beneath the sternum are best closed by suturing the anterior margin of the defect in the diaphragmatic muscle to the posterior sheath of the rectus muscle and to the anterior thoracic wall.

The most satisfactory material for closure of the opening is living suture of fascia lata removed from the thigh and stabilized in the tissues with silk. The round ligament of the liver can be incorporated in this closure to strengthen it as well as to re-establish its position on the anterior abdominal wall.

The closure of the large openings is facilitated by paralyzing the right side of the diaphragm by temporary interruption of the right phrenic nerve. This procedure, however, is not necessary in the closure of small openings. Preparation can be made to interrupt the phrenic nerve in the supraclavicular region after exploration of the opening and determination whether or not interruption is necessary.

Results:—Of the 534 patients having diaphragmatic hernia on whom I have operated, 14 had subcostosternal diaphragmatic herniae (foramen of Morgagni). All 14 patients underwent radical repair of the hernia through an abdominal approach. In 2 cases, an esophageal hiatus hernia was repaired at the same time. In 1 of these latter cases, the subcostosternal hernia was bilateral, with the pyloric end of the stomach extending into the right side of the thoracic cavity and the transverse colon into the left side. One of these was a recurrent subcostosternal hernia, the patient having had two previous operations elsewhere for repair of the hernia, one through a thoracic approach and one through an abdominal approach. The esophageal hiatus hernia which also was present consisted of herniation of about a fifth of the cardiac end of the stomach through an enlarged esophageal hiatus.

Of the 14 patients having subcostosternal diaphragmatic hernia, 6 were male and 8 were female. The hernia contained colon and omentum in 6 cases, omentum

only in 4, stomach, colon and omentum in 3, and stomach and omentum in 1. All 14 patients recovered from the operation. In the 14 patients operated on, there has been no recurrence of the hernia and all patients have been relieved of symptoms.

I should like to re-emphasize that the most important surgical consideration in this type of hernia is the difficulty of establishing a definite diagnosis of the hernia when the omentum is the only structure involved in the hernia. In some instances the shadow in the thorax may be interpreted as that of an intrathoracic tumor. One of these patients had undergone thoracoplasty previously, on the basis of a diagnosis of thoracic tumor. The condition was found to be a subcostosternal hernia which could not be reduced from the thoracic side. The patient was operated on later for repair of the hernia through an abdominal approach.

Herniae Due to Structural Defects of the Esophagus and Congenital Abnormal Esophageal Hiatus

There are several different types of herniation that occur through the esophageal hiatus. Some types are present at birth but are less common than other kinds of congenital diaphragmatic hernia; other types are not demonstrable until later in life and are the most common of all types of diaphragmatic herniae seen in adult life. I believe that most of this latter group of herniae are fundamentally of congenital origin and the result of abnormal development of the esophagus and diaphragm.

There are two principal types of congenital esophageal hiatus hernia which are due to congenital malformation of the esophagus. The first type results from the esophagus not elongating sufficiently to reach the diaphragm, so that various amounts of stomach remain suspended above the diaphragm. This is not a true diaphragmatic hernia, since the stomach has never been below the diaphragm. It is better termed a partial or complete thoracic stomach due to a congenital short esophagus. The second type results from a delay in the descent of the esophagus, and the esophageal hiatus is formed around the anlage of the stomach rather than the lower part of the esophagus, a mode of formation which results in an esophageal opening larger than normal. This occurs because the stomach descends behind the septum transversum in the early embryonic weeks and if its descent is abnormally delayed, the lumbar portion of the diaphragm will be imperfectly developed and the esophageal hiatus will be formed around the cardiac end of the stomach instead of around the esophagus. This will result in an abnormally large hiatus with deficiency both in the muscle ring and in the attachments of the diaphragmatico-esophageal membrane to the lower part of the esophagus. The degree of deficiency depends on the amount of gastric anlage in the thorax at the time of muscularization of the lumbar portion of the diaphragm. In most instances the esophagus continues to elongate normally but will result in imperfect fixation of the diaphragmatico-esophageal membrane to the esophagus and stomach and an abnormally large peritoneal fold which will extend well down on the cardia of the stomach. This abnormal relationship will permit much more flexibility of the esophagus in the enlarged hiatus than is normal. The greater the congenital deficiency in the formation of the esophageal hiatus, the more likelihood there is

that herniation of the stomach or other abdominal viscera will occur through it either before birth or later in life.

Symptoms:—In esophageal hiatus herniae which are present at birth, the symptoms may be abdominal and thoracic depending on the amount and type of abdominal viscera in the thoracic cavity. The thoracic symptoms associated with these herniae are rarely as marked as those associated with other cases of congenital diaphragmatic hernia, particularly such thoracic symptoms as dyspnea and cardiac irregularities. The thoracic symptoms produced by these herniae depend on whether or not more than one abdominal viscus has herniated into the thoracic cavity and the amount of increased intrathoracic pressure which is produced by them. In these herniae the stomach and the esophagus are usually the chief organs involved, and the symptoms are dependent on how much the normal function of these organs is interfered with and whether or not there is any mechanical obstruction to the esophagus or stomach, or associated spasm of

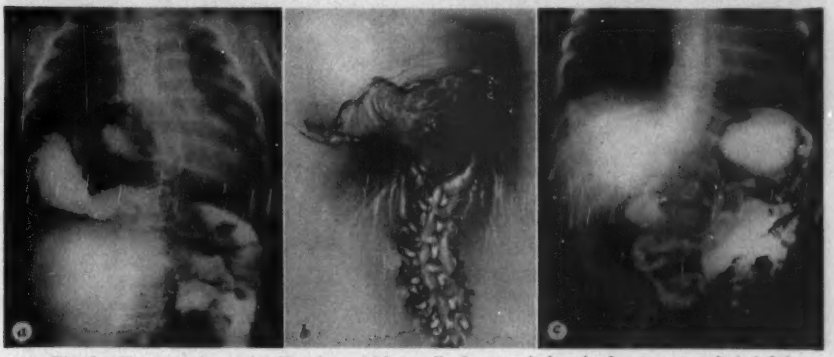

Fig. 5—Girl aged 6 months. Esophageal hiatus diaphragmatic hernia due to congenital deficiency posteriorly. *a*. Herniation of entire stomach and transverse colon into right side of thoracic cavity. *b*. Enlarged hiatus, defective posteriorly, repaired with silk and fascia lata. *c*. One month after abdominal repair of hernia. Stomach and colon in normal position below the diaphragm.

the lower part of the esophagus and the diaphragm (Fig. 5). There are usually varying degrees of dysphagia and vomiting. In some instances the infant may not be able to take anything by mouth and will require immediate surgical intervention, but in most instances this type of hernia is not associated with complete obstruction and the infant can carry on for varying periods of time by careful feeding.

In the esophageal hiatus herniae which occur later in life and are due to congenital malformation of the esophageal opening and in which the esophagus is of normal length, the symptoms are usually those related to progressive obstruction of the stomach and esophagus and may extend over many years. Inasmuch as these are sliding herniae, the symptoms are progressive and vary depending on the amount of viscera involved in the herniae as well as the degree of the distortion of the esophageal opening. Because of the slow, progressive character of these herniae, they rarely require immediate surgical intervention.

Surgical Treatment:—The management of esophageal hiatus hernia due to congenitally short esophagus presents a different problem from that of other types of esophageal hiatus hernia. In many of these cases the chief subjective symptom is dysphagia owing to the stenosis at the esophagogastric junction. This is best relieved by dilatations of the esophagus. Radical operative procedures should be carried out on those patients who are not relieved by conservative measures (Fig. 6).

In the operative treatment of these herniae, I prefer the abdominal approach.

The surgical problem in these cases is that of reconstructing the diaphragm over the elevated portion of the stomach: this can be accomplished if the esophagus is not too short. By complete and permanent interruption of the phrenic nerve the diaphragm usually can be elevated from 2 to 5 cm., and then by complete separation of the attachment of the esophagus from the attachments around the esophageal

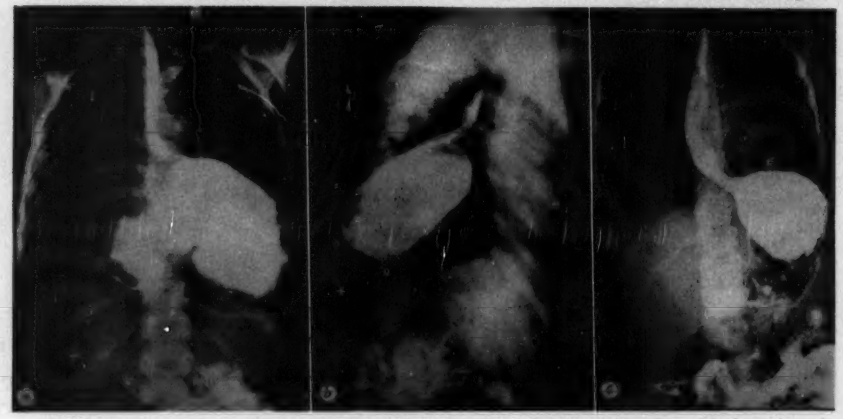

Fig. 6—Patient 5 years of age. Esophageal hiatus diaphragmatic hernia with intrathoracic stomach, partial shortening of the esophagus and herniation of transverse colon and loop of small intestine into posterior mediastinum. *a* and *b*. On admission, showing herniation of entire stomach and transverse colon into left lower part of thorax. *c*. One year after abdominal repair of the hernia. Entire stomach in normal position below the diaphragm. No symptoms. (From Harrington, S. W.: Diaphragmatic Hernia. In Nelson's Monographs on Surgery, 1950. In press.)

hiatus, from 2 to 3 cm. of the esophagus can be drawn down into the abdomen. The elevation of the diaphragm and the pulling down of as much as possible of the esophagus into the abdomen permit the esophageal hiatus to be closed around the lower end of the esophagus; by this means the esophageal hiatus is reconstructed at a higher level on the esophagus and the stomach is placed below the diaphragm. If the shortening of the esophagus is more than 5 to 6 cm. it may not be possible to obtain sufficient elevation of the diaphragm to enable one to reconstruct the esophageal hiatus above the stomach. Stricture at the esophagogastric junction is associated fairly often with congenital short esophagus as well as with other types of short esophagus. In most instances repeated dilatation of the stricture will relieve the obstruction. If obstruction cannot be relieved in these cases by dilatation, transthoracic resection of the esophagus may be required with reimplantation of the esophagus into the wall of the stomach.

There are many variations found in the position, and the amount of congenital deficiency, of the muscle of the ring of the esophageal hiatus which result in esophageal hiatus hernia, as well as in the attachments of the hernial sac to the stomach. The hernial sac, when present, is completely removed from its attachments to the stomach and the esophageal hiatus. The hiatus is then reconstructed around the lower end of the esophagus with stabilizing silk, and several layers of fascia lata are then interwoven between the overlapped margins of the hiatus. The lower end of the esophagus is sutured to the margins of this closure and to the diaphragm so as to prevent subsequent retraction of the esophagus above the diaphragm.

Results:—In 534 various types of diaphragmatic hernia which I have treated surgically, 426 were of the esophageal hiatus type. Of these esophageal hiatus herniae, 35 were classified as esophageal hiatus herniae with short esophagus. In 23 of these 35 cases, the short esophagus was considered the result of cicatricial contraction resulting from esophagitis and ulceration of the lower part of the esophagus.

In the other 12 cases the short esophagus was considered due either to actual shortening of the esophagus or to elevation of the esophagus as a result of malformation of the esophageal hiatus. In 1 of these cases, in which the patient was 16 years of age, there was malformation of the esophageal hiatus with the stomach and esophagus in normal position and herniation of the transverse colon through the esophageal hiatus into the posterior mediastinum. Eight of these patients were from 6 months to 14 years of age, 1 was 16 years of age and 3 were adults. Five of the patients were female and 7 were male. It is interesting to note that 2 of the children in this series were brother and sister. Both were operated on at 6 months of age.

All patients were operated on through an abdominal approach. The stomach and omentum were the only abdominal viscera involved in 9 cases; the stomach, colon and omentum in 2 cases, and in 1 case the omentum and colon were the only abdominal viscera involved.

In 8 of these cases phrenicotomy was done to produce permanent paralysis of the diaphragm and in 4 a temporary phrenic nerve interruption was performed by crushing the nerve. The esophageal hiatus was repaired and reconstructed at a higher level on the esophagus in all cases after reduction of the hernia.

All patients recovered from the operation. In 2 cases there was a slight protrusion of approximately 2 cm. of the cardia above the diaphragm, which has not progressed. All patients were relieved of symptoms.

Discussion

Dr. Roy Upham (New York, N. Y.):—As an officer of the National Gastroenterological Association and a member of the Program Committee, I want to thank Dr. Harrington for his presentation. I regret that due to a series of circumstances this morning I was only able to be present during his closing remarks. Dr. Harrington's presentation is the one particular paper on the program that I was most anxious to hear in its entirety.

Back in 1940 this extraordinarily brilliant doctor presented to the medical profession a 61-page monograph on diaphragmatic hernia, which has been the foundation of the entire subject. As the years have passed it seems that our distinguished speaker has evolved from the confusion and mass of symptoms some well-organized approach to the subject, both from a diagnostic point of view and a surgical point of view. He has stressed that none of the twenty-five different symptoms which usually occur are characteristic, but over the years presentations such as that of the short esophagus, published, I think, about twelve months ago, have developed the characteristic diagnostic signs of particular types of diaphragmatic hernia. Dr. Harrington has been extremely fortunate in the amount of material he has had to study and his contributions on diaphragmatic hernia have been the greatest of any surgeon in the world.

The establishment of the diagnosis of diaphragmatic hernia is parallel to that of the diagnosis of pancreatic disease. The physician, being continually mindful of these conditions, will, if constantly on the alert, detect cases of diaphragmatic hernia in his patients from time to time, being persistently "diaphragmatic hernia conscious".

A particular point in the diagnosis is to be stressed in the roentgen examination. It is insufficient to evaluate these cases with the fluoroscope alone. They will elude you. A series of films should be taken in every case.

Dr. Harrington, I am sure that the gentlemen present have profited immensely from your most valuable contribution.

THE CHOICE OF TREATMENT IN IDIOPATHIC DILATATION OF THE ESOPHAGUS*†

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and
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Dilatation of the esophagus regularly follows any continuing difficulty in the passage of food from the esophagus into the stomach. When this occurs without an obvious cause of obstruction, then this condition is properly called idiopathic dilatation of the esophagus. It produces a typical clinical syndrome, and many theories have been advanced to explain its etiology. The first hypothesis to gain widespread recognition, due to the influence of Mikulicz and Meltzer, was that the underlying cause was a spasm at the cardia. And although this concept of the etiology has been largely abandoned, the condition is frequently called cardiospasm in the American literature. The prevailing opinion at present regarding the etiology is the concept of Hurst, viz., an achalasia or failure of relaxation in the lower end of the esophagus. Changes in the ganglion cells of the myenteric plexus have been described in certain pathological specimens, and to such abnormalities in the vagus innervation of the constricted part of the terminal esophagus the achalasia has been ascribed. This is an attractive theory with much in its favor.

Clinical Groups

It can be shown that dilatation of the esophagus, without obvious cause, associated with the typical syndrome, falls into at least four separate categories, at least three of which have entirely different etiologies. These groups can be distinguished clinically.

1. *Cardiospasm:*—If spinal anesthesia to the level of T-6 is given to all patients presenting this syndrome most of the cases will show no significant changes in the dilatation of the esophagus. However, some of the cases will show a marked decrease in the dilatation and the barium mixture will pass promptly and freely into the stomach during the anesthesia. In the latter cases the cause of the dilatation is a true reflex cardiospasm. Marked clinical improvement will follow the removal of the irritative focus from which the reflex originates. Silent ulcers of the stomach or duodenum and cholelithiasis have been found as sources of such reflexes.

Theoretically of course, such a cardiospasm, reflex from another lesion in the abdomen, is no longer *idiopathic* dilatation of the esophagus. We want to emphasize, however, that until the results of the spinal anesthesia were known these cases had nothing to distinguish them either clinically or roentgenographically from those in the other groups.

*Read before the Fifteenth Annual Convention of the National Gastroenterological Association, New York, N. Y., 9, 10, 11 October 1950.

†From the Department of Surgery, The University of Rochester School of Medicine and Dentistry, Rochester, N. Y.

2. *Achalasia of the Cardia:*—Most of the cases probably fall into this group. Spinal anesthesia is not effective in significantly influencing the degree of dilatation. On esophagoscopic examination the cardia opens readily from the pressure of the end of the scope. The difficulty in food passing into the stomach is entirely functional. This is the achalasia of Hurst. It is difficult to understand why mechanical stretching of the cardia usually affords at least temporary symptomatic improvement, as this area does not seem to offer resistance.

3. *Partial Cardiostenosis:*—In a smaller number of the cases on esophagoscopic examination, although there is no scarring and the mucosa appears normal, a marked resistance to the opening of the cardia by the end of the scope or a bougie is met. Spinal anesthesia is also quite ineffective. These cases are found to have a hypertrophic musculo-fibrous band in the region of the cardia or a constricting band from the diaphragmatico-esophageal ligament. At operation

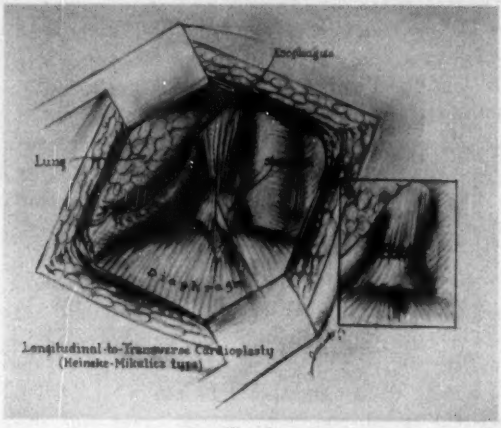

Fig. 1

the cardia or the esophagus at the point of the band will not freely admit the index finger. One of our first cases (Case 4, 1945) was a typical example of a partial stenosis[1] and we have had two other such cases. Kay found five patients in this group out of 17 operated cases[2].

4. *Dolichoesophagus:*—All cases of idiopathic dilatation of the esophagus have an increase in length as well as in width. One group of these cases have such an exaggeration of their length that the esophagus forms an S shaped curve with at least two separate fluid levels. These patients show no response to spinal anesthesia and on esophagoscopic examination have a nonresisting cardia if it can be reached by the scope, just as the achalasia group do. It may be, as Sweet[3] suggests, that this unusual length is merely a late form of the typical achalasia. Certain facts, however, suggest that this is not the explanation. In the first place it seems difficult to explain the development of an "S-shaped" curve with fluid pooling separately in the upper segment of the curve from a functional obstruction

at the lower end of the esophagus. Also such an unusual lengthening of the esophagus has been found in young infants. Furthermore, Freeman reported an amazing result in a patient with this type of megaesophagus. Immediate and permanent relief was reported as resulting from an esophagoplasty in the neck which shortened the esophagus. If the cause of the dilatation had been an achalasia at the lower end of the esophagus, it is difficult to understand how decreasing the length of the esophagus could relieve the syndrome.

Whether this dolichoesophagus group has achalasia as its primary etiology or not, it is desirable from the clinical standpoint to segregate these patients. After a simple cardioplasty the upper pouch of the curve still causes pooling with residual symptoms.

Treatment

The recognition of the clinical group into which the individual case falls is important as it influences the choice of treatment. In reflex cardiospasm the site from which the afferent arc arises should be found and eradicated, if possible. In

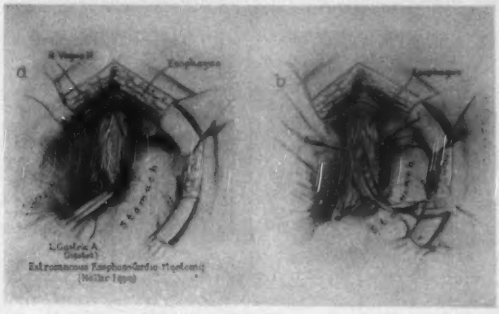

Fig. 2

the partial stenotic group mechanical dilatation of the cardia produces only temporary improvement and it is usually useless to carry out a long series of such dilatations. Cardioplasty should be performed ordinarily as soon as the clinical type is recognized.

Most of the cases fall in the group of achalasia. The severity of symptoms in a given patient in this group is frequently affected markedly by psychic factors and there are often symptomatic exacerbations and remissions due to the latter. Also there is an extreme range of severity in the symptoms of an individual case. Thus not only the clinical type but also the degree of its symptomatology should be appraised before deciding on treatment. In certain instances psychological support may be more important than physical measures. Mechanical dilatation of the cardia often affords temporary relief and may initiate a fairly long period of remission of symptoms. If this is the case, then it is reasonable to continue with periodic dilatation as indicated. We would suggest one condition to such a plan, even when symptomatic control seems fairly adequate. The roentgenographic appearance of the esophagus should also be checked periodically. If this shows

that the width of the dilated esophagus is gradually increasing then such dilatation is insufficient and more frequent dilatations or an operation is indicated to check the progress of the condition.

Operation

Although any individual series of operated cases is still relatively small the results of operation reported in the literature together with one's personal experience justify the following conclusions: (1) cardioplasty has a very low operative mortality; (2) adequate cardioplasty whether of the horseshoe (Finney) type or of the longitudinal-to-transverse (Heineke-Mikulicz) type will produce a free passage of food from the esophagus into the stomach, in the achalasia group and also in the cases of partial stenosis. In the cases of dolichoesophagus this will also improve the emptying of the lower segment of the curve but if nothing more is done there will still be pooling in the upper segment. Following such cardioplasty the width of the dilated esophagus diminishes but may or may not return to normal. There is marked symptomatic improvement but in very long standing cases there may be some residual dysphagia or phagophobia. In short then, the incapacitating syndrome of idiopathic dilatation of the esophagus can be largely or completely overcome by an operation carrying no undue risk.

We, therefore, feel that it is a mistake to defer operation unless dilatation and other conservative measures promptly control the symptoms. Most of these patients coming in to the surgeon today have had serious difficulty for five to fifteen years, and often have had as many as fifty dilatations. We recognize the value of dilatation in the treatment if it is followed by a large measure of symptomatic relief for several months. But we are convinced that where this procedure gives very ephemeral improvement or where the degree of enlargement of the esophagus proceeds in spite of it, then delaying operation merely prolongs the period of distress and disability without any compensating advantages. After experiencing the distressing dysphagia for several years some of these patients have an actual fear of eating which may linger on after operation, even when food readily passes into the stomach. Some psychiatrists feel that such a phobia may have been an initiating cause of the condition. Surely we agree that psychic factors can be important at least in aggravating the symptoms. However, we would urge consideration of the psychic trauma that occurs from the very real difficulty and discomfort these patients have to get sufficient food through the cardia to keep themselves alive. Such an experience extending over many years cannot contribute to their psychological normality.

Choice of Operation:—The primary object to be achieved by operation is to overcome the difficulty in the passage of food through the lower end of the esophagus. Here, as apparently in its counterpart megacolon, the dilated segment is not the chief seat of the difficulty but the disturbance is primarily in the function of the nondilated segment. Consequently, any plastic procedure permanently insuring the elimination of this segment and making a direct opening from the dilated esophagus into the fundus of the stomach should theoretically correct the dysphagia. A large measure of such relief has actually been found to occur

whether the cardioplasty was of the horseshoe (Finney) type (for illustration see Scott, 1945) or was achieved by a longitudinal incision closed transversely (Fig. 1). In either case, however, the incision must begin in the fully dilated segment and must extend through the cone of constriction well into the stomach. Recently the simple method of Heller, viz., the longitudinal division of all muscular and fibrous layers down to the intact mucosa (Fig. 2) has received an extensive trial by Maingot in London with excellent clinical results[4]. We can attest to the simplicity of this operation and to its excellent functional improvement (Figs. 3, 4 and 5).

The approach to the esophagus, whether it be through the abdomen or transthoracic, is usually relatively unimportant as the dilated esophagus, cone of constriction, and cardiac end of the stomach can be readily exposed from either approach. Most of the horseshoe type cardioplasties have been performed through the abdominal approach and most of the Heineke-Mikulicz type through the trans-

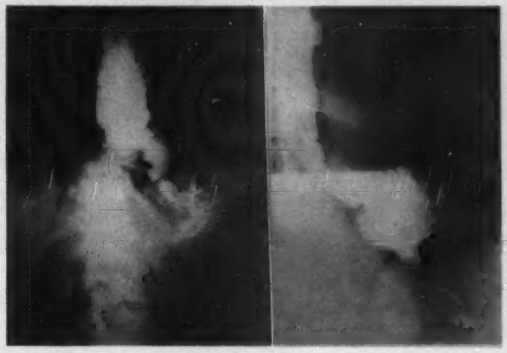

Fig. 3—F. B. Idiopathic dilatation of the esophagus achalasia type.
(A) Typical roentgenographic appearance.
(B) Spot film of the dilatation cone (2 cm. above cardia).

thoracic approach. The longitudinal myotomy (Heller type) can be performed from either approach but usually has been done transabdominally. If the dilatation of the esophagus is enormous (its diameter over one-third of the transverse diameter of the chest) then there is a definite advantage to the transthoracic approach as the wall of the boggy esophagus may be very friable. Also if the dilated esophagus is "S-shaped" with dual fluid levels (Dolichoesophagus) then also in our opinion it is preferable to use the transthoracic approach. In this group, if feasible, the esophagus should be mobilized, straightened and the upper fluid level eliminated.

Complications

Infection:—A few postoperative infections have been reported following cardioplasties. In the usual case with adequate preparation and precautions, particularly against the food debris in the dilated esophagus which most of these patients have when they enter the hospital, a postoperative abscess is extremely rare. In the long-neglected case with a boggy inflamed esophageal wall such an

infection is a definite danger. This, however, is not a valid objection to operation but instead is a cogent argument for early operation before the pooling of food has produced serious ulceration and infection throughout the esophageal wall.

Gastric Ulcer:—One of our patients developed an unusual complication. C. C., a young man of 24 had had dysphagia since he was aged 16 (8 years). The difficulty in swallowing fluid would start as a sensation of fullness in his throat and mid-way through his meal it would become marked. It was helped by standing and using the accessory muscles of swallowing. Carbonated liquids or coffee gave him a feeling of fullness very rapidly. Often to relieve the disagreeable fullness he would induce vomiting. Roentgenographic examination showed a typical dilatation of the esophagus ending in the usual smooth cone above the

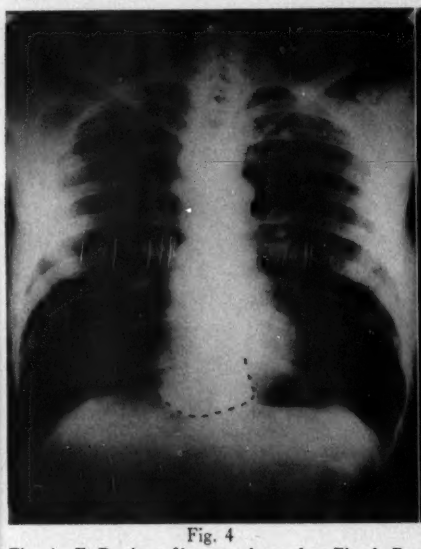

Fig. 4

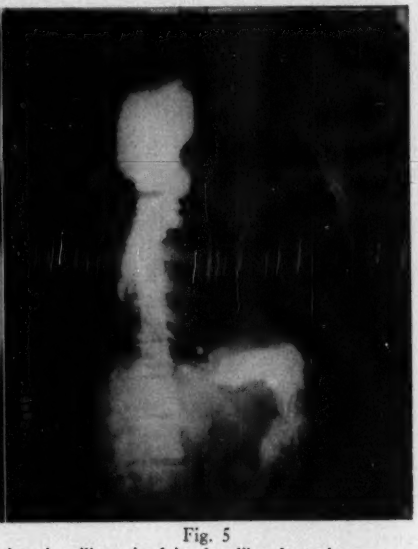

Fig. 5

Fig. 4—F. B. chest film two days after Fig. 3. Barium is still retained in the dilated esophagus.
Fig. 5—F. B. Postoperative results 1 year after esophagocardiomyotomy (Heller operation). Barium passes freely into the stomach. Dilatation of the lower thoracic esophagus is decreased though still present.

cardia. Due to the extent of the dilatation (over 10 cm. in diameter) it was decided to proceed immediately with operation after preparation rather than attempt mechanical dilatations. A transabdominal horseshoe type gastroesophagostomy was performed in May, 1946. This immediately relieved his dysphagia and his appetite returned. Two months after operation he began to have upper gastrointestinal bleeding and developed a marked secondary anemia. Roentgenographic examination showed excellent function at the anastomosis and was otherwise negative. Esophagoscopic examination also showed no source of the bleeding and an excellent anastomosis. On account of continued bleeding the stomach and anastomosis were explored. Just on the gastric side, the upper end of the anastomosis was densely adherent to the lateral leaflet of the crus of the dia-

phragm and there appeared to be a very small ulcer at this point on a dense scar tissue base. This was excised and the anastomosis at this point revised. Careful exploration of the remainder of the stomach revealed no other abnormality. A month following this operation he had severe recurrent bleeding. Repeated roentgenographic examinations revealed no ulcer until a year later when a well-marked ulcer niche was observed on the lesser curvature of the stomach about one inch below the anastomosis. On an ulcer regime the episodes of bleeding diminished but he developed increasing dysphagia from contraction of his gastroesophageal opening. In June, 1949 he was operated upon for the third time. A definite ulcer crater was found on the posterior wall of the stomach near the lesser curvature about 2 cm. below the anastomosis. The latter had been stenosed by dense scar tissue in which a main trunk of the left vagus nerve was embedded. The lower end of the esophagus, the cardia and the upper end of the stomach were removed and the stomach was anastomosed to the esophagus in the thorax. This completely relieved his obstructive symptoms but he still had some episodes of bleeding even after this radical procedure.

This patient developed a definite ulcer in the stomach shortly after his esophagogastrostomy. Such a complication is surely rare. However, we have seen one other patient whose original operation was done elsewhere with this complication. It is a serious one when it occurs. The question arose in our minds as to its cause. The ulcer was definitely in the gastric mucosa in both of these cases and, therefore, cannot be attributed to acid gastric contents regurgitated into the lower end of the esophagus. We wondered whether the involvement of a branch of the vagus plexus in the scar tissue at the point of anastomosis could have produced an irritative stimulation of the vagal nerve endings in the stomach, resulting in the formation of a gastric ulcer. We would suggest in the future that all vagal branches in the region of the anastomosis be divided to prevent their incorporation in postoperative scar tissue. If the extramucous esophagocardiomyotomy (Heller operation) is found to give lasting relief it would be preferable to either form of esophagogastrostomy and would probably eliminate the possibility of such incorporation of a vagus branch in scar tissue at the anastomosis. Maingot states that there are "no teasing complications such as peptic ulceration". We intend to give a thorough trial to this simpler procedure.

Conclusions

1. Four clinical types of dilatation of the esophagus without obvious cause are recognized viz.: (a) achalasia of the cardia, (b) true cardiospasm, (c) partial cardiostenosis and (d) dolichoesophagus.

2. The clinical type influences the proper treatment.

3. Conservative treatment including dilatation should be used only where it fairly adequately controls the symptoms.

4. Esophagogastrostomy in the uncomplicated case has a small operative risk and usually adequately relieves the dysphagia.

5. A postoperative gastric ulcer is an unusual but serious complication of esophagogastrostomy. Suggestions of technic are made to attempt to avoid this unfortunate sequela.

REFERENCES

1. Scott, W. J. M.: Idiopathic Dilatation of the Esophagus. Ann. Surg. **122**:582-605, (Oct.), 1945.
2. Kay, E. B.: Surgical Treatment of Cardiospasm. Ann. Surg. **127**:34, 1948.
3. Sweet, R. H.: Idiopathic Dilatation of the Esophagus. S. Clin. North America. **27**:1128, (Oct.), 1947.
4. Maingot, R.: Surgical Treatment of Cardiospasm. Postgraduate Med. **5**:351-360, (May), 1949.

Discussion

Dr. David J. Barry (New York, N. Y.):—It is worthwhile only to emphasize first that the very real and logical distinction between the types of dilatation of the esophagus which Dr. Scott has made, is most helpful in understanding and treating these cases as they occur.

There is only one question which I should like to ask Dr. Scott, and perhaps he can salvage the time that remains by adding to the information he has already handed us, and that is, how frequently does dilatation of the esophagus occur as a reflex from cardiac or aortic trouble, since we can't seem to keep the heart and the esophagus separated, this morning.

I have a case in mind that troubled us for a long period, at one time, because the man came to the hospital with a six-year story of angina. He had an acute myocardial infarction, and during a period of about six weeks frequent x-ray showed this large shadow in the mediastinum, which turned out to be a dilated esophagus, and the symptoms were very hard for us to explain, and the patient was in the greatest amount of distress, being unable to take food.

Ultimately, however, as the cardiac status became worse, and the man went into congestive failure three months afterward, a study of the esophagus at that time revealed none of the spasm of the cardia or dilatation of the esophagus, so we called that a reflex cardiospasm, but, being the only one we had seen associated with myocardial infarction, I wonder if such things occur frequently.

Dr. Edwin Boros (New York, N. Y.):—The diffuse dilatation of the lower end of the esophagus which characterizes cardiospasm is an aftermath of a pinching at its lower end. This constriction does not take place at the cardia, as originally suggested, but is due to functional failure at this point.

Microscopic degenerative changes in Auerbach's plexus have been demonstrated with final disappearance of the ganglion cells in the wall of the lower esophagus. To this alteration of structure, the condition known as idiopathic dilatation of the esophagus or achalasia is most generally attributed. That a nutritional factor may be of more than speculative value was emphasized by Etzel, whose figures yielded a high incidence of patients with cardiospasm in districts where pellagra was prevalent; so that a vitamin deficiency state may well be at the background of this condition. Diffuse spasm of the gullet has likewise been known to occur as a reflex manifestation of abdominal disorders such as gallbladder involvement. That both the vagi and sympathetic nerves play a definite

role in the control of the opening and closure of the lower end of the organ is well recognized.

It is apparent therefore that in considèring the means of therapy for cardiospasm, possible etiologic factors require proper perspective and balance, and the mode of approach is guided in great measure by the presence or absence of ascertainable findings. Local esophagitis, an unhealed ulcer, be it peptic or otherwise, local stricture formation, may be the bases of a diffuse esophageal dilatation; moreover, carcinomatous degeneration in a long-protracted state of esophageal stagnation has been observed as a complication by the speaker.

Accordingly, first and foremost, before the institution of treatment, be it conservative or surgical, is the establishment of a proper diagnosis. This cannot be completed without esophagoscopic exploration. The simplicity and safety of the procedure has been demonstrated.

Peptic ulcer or gallbladder disease, when encountered, present no special therapeutic problem. Where no specific findings are apparent, dilating bags and bougies have served well in affording the patient relief from symptoms.

The author has seen no satisfactory response whatsoever in the use of atropine, antispasmodics in general, nitrites, etc.; furthermore, the paradox of improved symptomatology without paralleled diminution in size of the organ, stands as a phenomenon which characterizes the condition. With chronicity and scar formation, stricture may ensue. Often dilatation can be accomplished by the passage of bougies passed through an esophagoscope. Surgery may be required in cases of neglect and unyielding stenosis.

There are few things more gratifying than the satisfaction of one afflicted with dysphagia who, with the proper institution of prompt therapy, is afforded that long-lost comfort of normal degiutition, of which he was so summarily deprived.

Dr. Horace W. Soper (St. Louis, Mo.):—I wish to make a very brief practical remark on the subject of dilatation of the cardia, or cardiospasm, whether it is a spasm or merely achalasia. In the dilatation, very early in my clinic we used a large rubber dilator. There was a patient from out of town and we thought we would hurry up the thing by giving her a daily dilatation. She had three dilatations. She started home the next day, had a break and died of peritonitis.

Since that time we have allowed one week to elapse before a second dilatation —always a week between dilatations.

We have also used the small Einhorn dilator, which is much more effective than the larger type we formerly used.

Dr. W. J. Merle Scott (Rochester, N. Y.):—I want to thank the discussers for their kind remarks.

I am very glad that someone spoke about the medical side of the treatment, because I don't feel at all competent to speak of that. I think there is a nutritional factor in many of these patients, whether it is primary or secondary, and the nutritional factor should always be overcome insofar as possible by the use of vitamins, etc.

I call your attention to the assistance of the esophagoscopist. We elicited his aid both in the diagnosis, to determine the exact state of affairs, as I think I showed you in my paper, and also to help us clean out the esophagus. If you get the esophagus well cleaned out of food residue before operation, then you have no real fear of any serious infection as a complication of your operation.

The antispasmodics, I agree, have not been of great use in our medical clinic among our colleagues whom I have seen try to handle these cases in that way, or in preparation for operation. Possibly with the newer antispasmodics that are coming along, we may find that there is such a drug therapy eventually. But if our idea of the etiology in the achalasia group as being due to an absence of myenteric plexus ganglion cells, and their connections in the wall of the esophagus is correct, then I doubt whether the drug therapy, even replacement of a vagotonic effect, will give you the coordination that you need to overcome the achalasia.

I am speaking in this presentation of the 20 to 25 per cent of the cases that are not satisfactorily handled at the present time by medical measures. Also I think that as the knowledge of the excellent result attained by operation is more generally spread, you will find that there will be a little increase in that figure. In other words, there will be borderline cases who can be carried along by conservative measures, but somewhat unhappily both to the patient and to his physician, that will be better off operated upon. But the number, I believe, will not be above 35 or 40 per cent even with these borderline cases.

CHRONIC URTICARIA ASSOCIATED WITH HYPOCHLORHYDRIA OR ACHLORHYDRIA*

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The association of urticaria with low gastric acidity has been previously recognized, but too infrequently utilized for the benefit of the patient. Hajos[1,2], in a study of chronic urticaria, found the hydrochloric acid content of the stomach to be absent in 65 per cent of the patients tested and lowered in 30 per cent; favorable results were obtained by him with the use of hydrochloric acid and histamine in the treatment of urticaria. Bray[3] suggested hydrochloric acid as a form of therapy in allergy, using large doses over long periods of time. It had been postulated by various writers[3,4] that, with the absence of hydrochloric acid in the stomach, there might be absorption of incompletely split protein, which could possibly account for sensitivity reactions in patients with urticaria, asthma, and in some others with angioneurotic edema and certain forms of eczema.

During the past fifteen years we have observed many patients with chronic urticaria. Some had had urticaria for several years and had consulted us for treatment for arthritis. They usually complained of gas, abdominal distention, "indigestion" and abdominal discomfort. Their first thought was that they had had too much acid and the usual array of alkalis being handy, they had taken one or more doses of various alkalis after meals. They also complained of fatigue, of being mentally dull, and as they expressed it, they "felt run down". The "run down" feeling may be accounted for by the fact that, without free hydrochloric acid in the stomach, the patient does not absorb Vitamin B, and thus develops a B avitaminosis. Many had seen several physicians and had had many tests, including allergy. A great many had also had various forms of treatment, including periods of rest, sedation, elimination diets, intravenous calcium, alkalis, colonic irrigations, etc. In spite of the galaxy of tests, it is surprising that so few had had a gastric analysis.

Materials and Methods

A test meal of 200 c.c. of 7 per cent ethyl alcohol was used. The nasal passage is anesthetized and a small Levine or Weiss tube is passed through the nose into the stomach. A fasting specimen is taken and further specimens every fifteen minutes for one hour after the alcohol meal is given, and every thirty minutes for the next hour or longer if the peak is not passed. If there is no free hydrochloric acid, histamine stimulation is used to determine if it is a true or relative achlorhydria. The patients included in this study had urticaria of long standing, six months or more, and the cause of the urticaria could not be determined. Those classed as

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hypochlorhydria had less than 20 degrees of free hydrochloric acid in any specimen. Dilute hydrochloric acid in liquid or capsule form was administered to those patients with achlorhydria or hypochlorhydria. The largest dose was given with the largest meal. Acute urticaria is not included in this study. The following case report is illustrative of the cases under discussion.

H. C., a male, age 35, was first seen in 1937. His chief complaint was urticaria of two years' duration. He had been seen by an allergist and several internists. He had had a complete gastrointestinal x-ray study, including colon and gallbladder, also cultures of nose and throat and feces, blood chemistries, etc., and x-ray of all teeth. The allergist had performed the usual series of tests, and obtained reactions to certain foods. He had eliminated all the foods suggested by the allergist, was on sedatives, tonics, alkalis, etc., all without result. His internist thought the urticaria might be due to business tension and advised him to go to Florida for a month's rest, again without any result, in fact he seemed to be worse while in Florida. One of us (W. B. R.) saw him shortly after his return from Florida and at that time there was urticaria involving the entire body with occasional angioneurotic edema of the lips. The general physical examination was normal throughout. The gastric analysis (the only test performed) showed: No free hydrochloric acid, highest total acidity—10 degrees. He was started on dilute hydrochloric acid and within a few days his urticaria began to lessen and within a month had almost disappeared except for an occasional mild attack of no particular consequence. At intervals he had discontinued the acid and after a short time the urticaria returned, but on resuming the acid the urticaria again disappeared. This has continued for thirteen years with almost complete relief from his urticaria.

Analysis of Forty Cases

The patients with achlorhydria obtained the best results. Twenty-two, or 55 per cent, had an absence of free hydrochloric acid. Eighteen, or 82 per cent, in this group were almost completely relieved. Two, or 9 per cent, were partially relieved, that is, there were times when they were symptom-free and at other times the symptoms were less severe. These patients were much more comfortable than before beginning the dilute hydrochloric acid. Eight, or 20 per cent, had hypochlorhydria and four, or 50 per cent of the eight obtained almost complete relief. Two, or 25 per cent, obtained partial relief. In the forty cases studied, twenty-two, or 55 per cent, were almost completely relieved and four, or 10 per cent, obtained partial relief, making a total of 65 per cent that obtained sufficient relief to make these patients comfortable, whereas they had been unable to obtain relief from any other form of treatment. This, we believe, is well worthwhile.

Discussion

When a patient presents the symptoms of flatulence, gas, indigestion, fatigue, mental dullness, etc., without definite pain and with a history of obtaining only partial relief from alkalis, one should suspect either a hypochlorhydria or achlorhydria. These patients frequently have had gallbladder and gastrointestinal

x-ray series with negative or, at the most, minor findings. Because of the absence of x-ray evidence of organic disease, they are told they have nothing wrong, that their symptoms are due to nervous tension, etc., or that they have a gastrointestinal allergy. In an effort to obtain relief they go from physician to physician. X-rays and tests are repeated and still their symptoms continue. In our experience it appears that the symptoms of hypochlorhydria and achlorhydria are not sufficiently stressed and are too infrequently recognized. It also appears that a gastric analysis is not done as often as it is indicated and not very often in the average practitioner's office. Many complicated laboratory procedures are done while a fractional gastric analysis, a comparatively simple procedure, is neglected. We cannot account for this neglect unless it is the patient's objection to the passing of the tube and the physician's failure to insist upon it because of his lack of knowledge of the proper technic. We think that more care should be taken in passing the tube, so that one does not frighten or upset the patient to the extent that they will always think and speak of it as a horrible experience. If one observes some attempts to pass a tube one can understand why the patient might object to having the test done, and perhaps why some physicians do not like to do it. It may seem academic to discuss such a simple process as passing a tube, but there is a real need for a better appreciation of what a proper technic can mean to a patient. We can recall many patients who at first objected to having a gastric analysis because some friend had referred to her own test as a "horrible experience". The nasal passage should be anesthetized using a nasal applicator, first being careful to express any excess material. A small Levine or Weiss tube should be used and it should not be forced. It should be held lightly between the thumb and index finger merely as a guide. If the patient begins to cough and wheeze, the tube should be withdrawn a short distance, the patient asked to breathe deeply through the mouth, the tube passed gently, moving only a short distance each time the patient swallows. If the tube is held as above described one can feel the tug as it slips down the esophagus, and if this tug is absent it is a sign the tube is curling up somewhere along the passage. If the patient has difficulty, one should not try to force the tube down. You must take your time and if you are careful you can always pass the tube. At times it may be necessary to give the patient one or two swallows of water. This does not seriously affect the analysis and may save the physician considerable trouble and the patient a great deal of discomfort. In twenty years experience, and in passing hundreds of tubes, we have failed only once and this patient would not let us try.

It is not our intention to suggest that all patients with chronic urticaria have hypo- or achlorhydria, but we do believe that it is sufficiently frequent to warrant a gastric analysis of all these patients. Conversely, it does not hold that everyone with hypo- or achlorhydria develops urticaria and we do not know why some develop urticaria while others do not. There are a great many other symptoms produced by hypo- or achlorhydria that one should recognize if one is observant. We would be doing a service to our patients if more gastric analyses were done.

Conclusions

1. Chronic urticaria is frequently associated with hypo- or achlorhydria.

2. A gastric analysis is a simple office procedure and should be done more often.

3. The case report included is a typical example of cases observed by us.

4. Our cases were those that had been proven not to be due to allergy or any other known cause and were usually classed as chronic urticaria of unknown origin.

REFERENCES

1. Wise, F. and Salzberger, M. B.: 1938 Year Book of Dermatology and Syphilology, p. 151.
2. Perry, C. Baird: New England J. Med. **224**:649-658, (April 10), 1941.
3. G. W. Bray: "Recent Advances in Allergy", second edition. T. & A. Churchill, London. Quart. J. Med.
4. Gray, I. and Walzer, M.: Am. J. Digest. Dis. & Nutr. **3**:403-404, (Aug.), 1936.

Discussion

Dr. Samuel Berger (Cleveland, Ohio):—The authors present an interesting observation concerning the relationship of hypo- or achlorhydria in the production of chronic urticaria.

In achlorhydria we may presume or presuppose the presence of an abnormal gastric mucosa secreting an insufficiency of hydrochloric acid, and there may be a complete absence not only of hydrochloric acid but also of pepsin; thus proper initiation of the digestion of protein fails to take place; therefore incompletely split proteins or foreign proteins enter the circulation and become allergens in previously sensitized individuals, causing the release of histamine or a comparable substance which can produce wheals.

The gastroenterologist finds hypo- or achlorhydria frequently, but the presence of chronic urticaria rather rarely. This is very fortunate for these achlorhydrics to be spared that most annoying and distressing condition. It is equally fortunate for the gastroenterologist or internist to be spared the embarrassment of failure to relieve this very trying malady not only after the administration of adequate amounts of hydrochloric acid but even after having tried everything known, both externally and internally.

In completely and well-studied instances of chronic urticaria, there still remains an undetermined or X factor, related probably to the central nervous system, a possible sympathetic imbalance. I cite the following example: A very prominent and very active attorney, about sixty years of age, had a total gastrectomy for carcinoma of the stomach. Shortly thereafter he developed urticaria, which became persistent and extremely troublesome, for more than a year.

I have known him for twenty years and I have known that he had a complete achlorhydria and an atrophic gastritis, yet not until, and at no time before, his gastrectomy did he develop urticaria. The disturbed emotional state, to say the least, contributed to the mechanism which resulted in chronic urticaria.

All of us see subtotal or total gastrectomies, with hypo- or achlorhydria, but chronic urticaria has not been noted as a feature.

Hypo- and achlorhydria are extremely common, especially in the aged, but, again, fortunately, chronic urticaria is infrequent. Further evidence is needed to

prove a causal relationship of hypo- or achlorhydria in the production of chronic urticaria.

Dr. Harry M. Eberhard (Philadelphia, Pa.):—In my office we see many cases of achlorhydria and hypochlorhydria and have noted a great number that have associated chronic urticaria. I have been impressed that in a great number of cases the average man in general practice fails to do a fractional gastric analysis or to refer the patient to someone who would do it for him. As Dr. Rawls has stated, unless a gastric analysis is done one cannot expect much success. Of note, it has been my experience that when many patients have come to my office complaining of "burning in the stomach" no free hydrochloric acid was noted and frequently the total acidity had been extremely low. These patients had been given bicarbonate of soda over a long period of time, which of course was contraindicated and in many cases had made the patient distinctly worse. I would like to add that when we see a patient with either achlorhydria or hypochlorhydria that is not histamine refractory we always check the concentration of rennet and pepsin and find it invaluable in directing treatment.

May I compliment Dr. Rawls for presenting this very valuable paper.

Dr. William B. Rawls (New York, N. Y.):—I should like to say in my closing remarks that we did not wish to suggest that all patients with urticaria would have an absence of hydrochloric acid or have a hypochlorhydria, but that we did wish to emphasize that every patient who has a chronic urticaria should have a gastric analysis, because we found 65 per cent or over of our patients with chronic urticaria did have a hypochlorhydria or achlorhydria and that 65 per cent of this group obtained relief when given hydrochloric acid.

We all know that we have a lot of hypochlorhydrias or achlorhydrias, many of whom do not have urticaria, but they have the symptoms as I suggested above. I should like to emphasize once again that a large percentage of these people do have hypochlorhydria or achlorhydria, and when you give hydrochloric acid, 65 per cent get relief and this in patients that have made the rounds for several years, in my opinion, is well worthwhile.

GASTROINTESTINAL FUNCTION FOLLOWING RADICAL PANCREATICODUODENECTOMY*†

CHARLES B. RIPSTEIN, M.D.
and
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In recent years, it has become possible to apply the principles of radical cancer surgery to neoplasms of the head of the pancreas. Following the pioneer work of Whipple[1] in 1935, the operative procedure has become well standardized and can now be carried out with an acceptable mortality.

The technical difficulties of removing a tumor in this region are numerous. Many important structures are liable to injury and because of this it is often impossible to fulfill the requirements of adequate excision. Many authors[2,3] have indicated that the curative value of the radical operation is doubtful in carcinoma

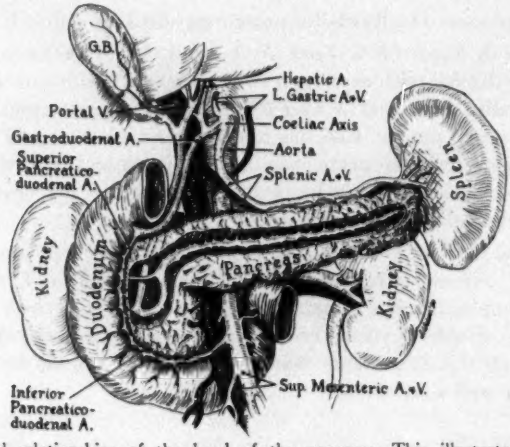

Fig. 1—Anatomical relationships of the head of the pancreas. This illustrates the technic difficulties involved in pancreaticoduodenectomy.

of the pancreas and that the relief of biliary obstruction offers maximum palliation with minimal mortality. Child[3] feels however, that since no other procedure offers any possibility of cure, radical resection should not be abandoned but rather extended to the point of removing the superior mesenteric vein and the entire pancreas.

Our own experience with this operation has not been too satisfactory from the standpoint of cure. In 20 cases of carcinoma of the pancreas, radical

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resection has been possible in 8 with 1 operative death. The average survival in those cases recovering from surgery was 14 months. Twelve cases were treated by short-circuiting the biliary obstruction and their average survival was 6 months. This is not a fair comparison because the unfavorable cases with hepatic metastases were all in the latter group. It appears likely that in most instances a simple cholecystenterostomy would offer as long a period of palliation as would pancreaticoduodenectomy. Such an attitude, however, would remove all hope of cure and consequently radical resection is fully justified if the diagnosis can be definitely established. This may offer a great deal of difficulty, even with the aid of frozen section examination of biopsies obtained at the operating table. A positive biopsy establishes the diagnosis beyond doubt, but a negative biopsy cannot be relied upon to rule out carcinoma. We feel that the radical operation should only be used in cases in which the diagnosis is clearly established clinically or by pathological examination.

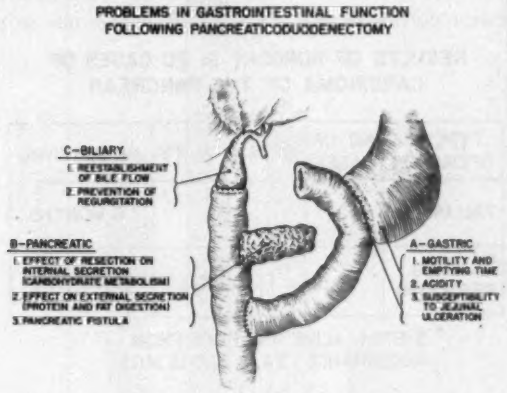

Fig. 2

In this series, two cases have been subjected to a two-stage procedure and six have been resected in a one-stage operation. The only fatality occurred in the second group and was due to a massive gastrointestinal hemorrhage on the sixth postoperative day (no autopsy was obtained). We feel that a single-stage operation is preferable because the technical difficulties imposed by vascular adhesions make the second stage an extremely hazardous venture.

Restoration of the gastrointestinal tract following resection of the duodenum and head of the pancreas, presents many problems. Pearse[4] has advocated that a standardized procedure be adopted and he stresses the following points:

1. Retrocolic end-to-side gastrojejunostomy.
2. Implantation of the common bile duct into the intestine.
3. Anastomosis of the pancreas to the intestine.
4. The use of a defunctionalized loop to divert the gastric contents from the biliary tract.

A great deal of controversy has been aroused concerning the merits of various methods of biliary and pancreatic anastomosis but few careful post-operative studies of gastrointestinal function have been reported. We have recently had the chance to study five cases in which preoperative pancreatic enzyme studies had been carried out. This presented a unique opportunity to assess the effects of the surgical procedure, and the results of this investigation form the basis of the present paper. It is most convenient to consider the various organs involved in reconstruction of the gastrointestinal tract and attempt to point out the complications which may arise in their function following resection of the head of the pancreas. In this way we can best evaluate the methods of obviating such complications.

Stomach

In most of the suggested operations for carcinoma of the pancreas, very little of the stomach is resected. This would appear to be a disadvantage because removal of the major portion of this organ would enable one to perform a more

RESULTS OF SURGERY IN 20 CASES OF CARCINOMA OF THE PANCREAS

TYPE OF OPERATION	NO. OF CASES	OP. MORTALITY	AV. SURVIVAL
PALLIATIVE	12	0	6 MONTHS
* RADICAL RESECTION	8	1 (12.5%)	14 MONTHS

* 3 STILL ALIVE AND FREE FROM RECURRANCE : 24,16 AND 12 MOS.

Fig. 3

radical excision of the pancreatic lesion. In addition, an adequate subtotal gastrectomy helps to prevent jejunal ulceration. Owens[5] has pointed out that the danger of such a complication exists because the situation is comparable to that in the Mann-Williamson dog, in which the alkaline duodenal contents are diverted from the anastomosis. He recommends that to avoid stomal ulcer, the biliary and pancreatic anastomoses be placed proximal to the gastroenterostomy so that the stoma will not be exposed to undiluted gastric juice. We prefer to resect sufficient stomach to produce achlorhydria, since in our experience jejunal ulceration never occurs in the absence of free HCl. Fig. 5 shows the gastric acidity values in 5 cases. In all, the free HCl has been reduced to zero from a normal preoperative level. Gastric motility has been normal in all cases and with the Hofmeister type of anastomosis the stomach retains food for 1-2 hours. We feel that either an anterior or a posterior anastomosis is perfectly satisfactory. We prefer the former because it is easier and less complicated and there appears to be no reason to change this opinion.

Biliary Tract

Reestablishment of the biliary flow into the intestine is of paramount importance. Without bile, fat digestion is impaired and vitamin deficiencies result from inadequate absorption. It has been frequently stated that anastomosis of the common duct to the jejunum is to be preferred to cholecystjejunostomy because of the lessened danger of biliary fistula and ascending cholangitis. Our experience does not agree with this. The use of the gallbladder is technically much easier and there is less likelihood of stricture at the anastomosis. If a defunctionalized Roux-Y loop is used, the incidence of cholangitis is extremely low. In only one case, have we been able to demonstrate regurgitation of ingested

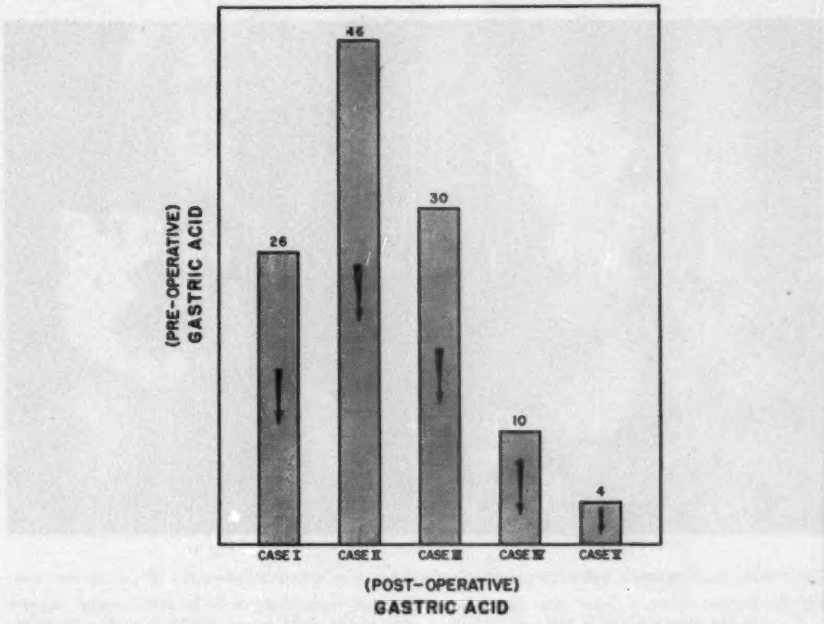

Fig. 4—Pre- and postoperative gastric acidity levels in 5 cases of radical pancreaticoduodenectomy.

barium into the gallbladder, and in no instance was barium seen in the bile ducts. There have been no clinical cases of cholangitis and postoperative biliary drainage has always revealed clear normal bile containing no pus cells or detritus. The danger of biliary fistula can be forestalled by closing the end of the common duct with two inverting layers of fine silk sutures. In the past, this dangerous complication has always occurred following ligature of the duct, but to date, we have not observed it after suture closure. Liver function has remained normal throughout the period of survival in all cases, and there have been no instances of recurrent jaundice.

Pancreas

It has been clearly established in the past that excision of the head of the pancreas does not disturb the endocrine function of the gland. This has been confirmed in our studies and none of these patients have shown any derangement of carbohydrate metabolism.

In the early cases of pancreaticoduodenectomy, no attempt was made to preserve the exocrine function but more recently it has been recognized that this is technically feasible and physiologically desirable. The two methods of anastomosis used in these patients have been end-to-end and end-to-side implantation of the cut end of the pancreas into the defunctionalized jejunal loop. The

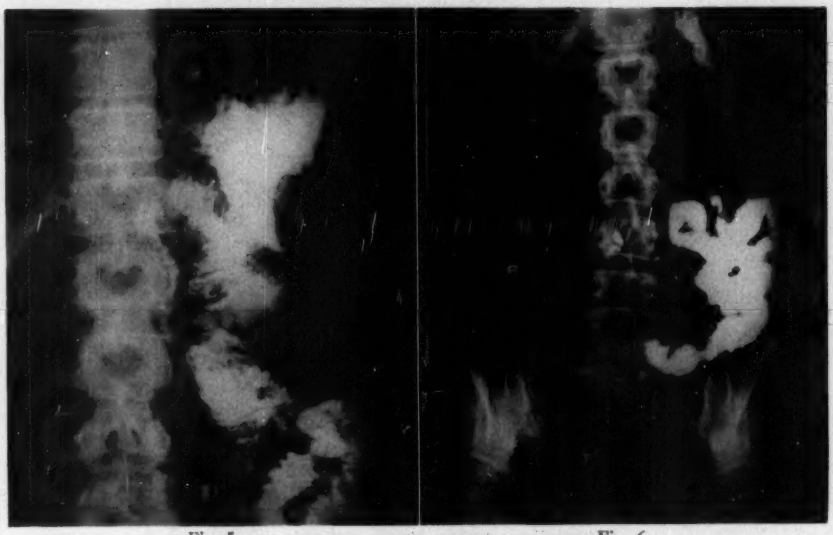

Fig. 5 Fig. 6

Fig. 5—X-ray of stomach following pancreaticoduodenectomy showing the extent of gastric resection.

Fig. 6—Barium x-ray, 1 hour after ingestion of barium. Note that there is still residual barium in the stomach and a trace of barium is seen in the right upper quadrant, indicating some regurgitation through the defunctionalized loop.

results have been similar with both methods and they conclusively demonstrate that it is possible to preserve the digestive function of the pancreas. It is interesting to note, that in all cases, pancreatic amylase and lipase have been low or absent from the duodenal drainage preoperatively. This phenomenon may be of some value in diagnosis. In every instance, the enzyme content of the pancreatic juice has returned to normal following excision of the tumor and pancreaticojejunostomy. Examination of the stools has shown normal fat and protein digestion and we have encountered no instances of steatorrhea.

The following brief case reports illustrate the changes produced in gastrointestinal function by pancreaticoduodenectomy.

Case 1:—P. F., male, age 44. Duration of symptoms 3 months. Loss of weight, diarrhea, jaundice. Survival after radical resection, 12 months.

	Preoperative	*Postoperative*
Gastric Acid	Free 26 Total 45	Free 0 Total 10
Duodenal Amylase	0	17
Duodenal Lipase	0	1.6
Blood Lipase	3.6	2.1
Blood Amylase	84	113

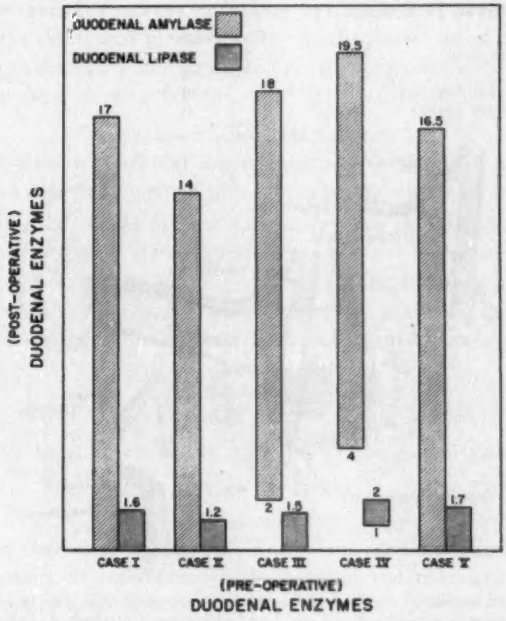

Fig. 7—Duodenal enzyme content before and after radical pancreaticoduodenectomy.

Case 2:—D. S., female, age 44. Duration of symptoms 3 months. Jaundice, weight loss, weakness. Survival 7 months.

	Preoperative	*Postoperative*
Gastric Acid	Free 46 Total 78	Free 0 Total 18
Duodenal Amylase	0	14.0
Duodenal Lipase	0	1.2
Blood Lipase	4.0	1.8
Blood Amylase	70	108

Case 3:—R. B., male, age 62. Duration of symptoms 9 weeks. Jaundice, weight loss. Survival 22 months.

	Preoperative	*Postoperative*
Gastric Acid	Free 30 Total 50	Free 0 Total 20
Duodenal Amylase	2	18
Duodenal Lipase	0	1.5

Case 4:—B. E., female, age 54. Duration of symptoms 6 weeks. Diarrhea, weight loss, jaundice. Survival 8 months.

	Preoperative	*Postoperative*
Gastric Acid	Free 10	Free 0
	Total 24	Total 8
Duodenal Amylase	4	19.5
Duodenal Lipase	1.0	2.0

Case 5:—C. F., male, age 60. Duration of symptoms 6 weeks. Jaundice, weight loss. Survival 14 months.

	Preoperative	*Postoperative*
Gastric Acid	Free 4	Free 0
	Total 18	Total 6
Duodenal Amylase	0	16.5
Duodenal Lipase	0	1.7

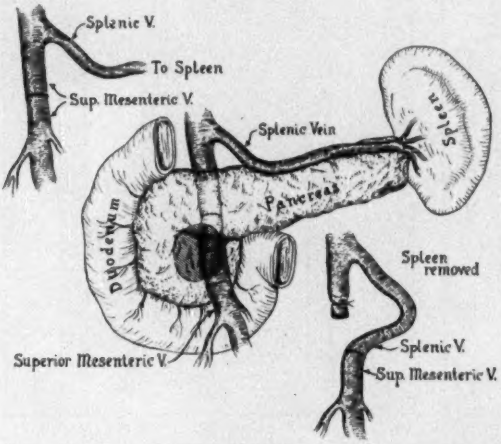

Fig. 8—An example of segmental resection of the superior mesenteric vein with end-to-end anastomosis. This procedure was carried out in Case 2 and illustrates a method of obtaining a more radical resection.

Comment

From this rather limited experience, several conclusions may be drawn. Resection of the duodenum and the head of the pancreas can be carried out with an acceptable mortality rate. Furthermore, the gastrointestinal tract may be reconstructed so that it functions in a fairly normal manner. Unfortunately, the end results in terms of cure have been far from satisfactory. The weak point of this operation lies in the necessity for preserving the superior mesenteric vein. This vessel passes through the substance of the pancreas and is usually in close proximity to the tumor. Child[3] has indicated that it is often possible to tie off the portal vein with impunity but it is difficult to be sure that the collateral circulation will be adequate in any given case. We have seen instances in which infarction of the bowel occurred following damage to this vessel. He has also pointed out that it is feasible to anastomose the superior mesenteric vein to the splenic vein

or the inferior vena cava and sacrifice that portion of the vessel which traverses the pancreas. We have been able to confirm his experimental results in dogs, and in one human case, we have successfully excised a segment of the vein involved by the growth and reconstituted the vessel by end-to-end anastomosis. This patient (Case 2) survived 7 months before dying from recurrence. The mesenteric vein was patent at postmortem examination.

The ideal operation for carcinoma of the head of the pancreas would involve resection of the entire pancreas including the superior mesenteric vein and anastomosis of the distal end of the vein to the proximal end of the splenic vein. With earlier diagnosis and a more radical operative procedure, we may expect to obtain better results than at present.

Summary and Conclusions

1. The operation of pancreaticoduodenectomy is the only procedure which gives any hope of cure in carcinoma of the head of the pancreas.

2. At present, the results are not satisfactory but the operation can be made more radical by removal of the entire pancreas and the superior mesenteric vein.

3. It is possible to maintain the normal function of the gastrointestinal tract following excision of the duodenum and head of the pancreas. Pancreatic enzyme secretion can be restored to a normal level and no deficiencies of digestion or absorption exist.

REFERENCES

1. Whipple, A. O., Parsons, W. B. and Mullins, C. R.: Ann. Surg. **102**:763, 1935.
2. Cattell, R. B. and Pyrtek, L. Z.: Ann. Surg. **129**:840, 1949.
3. Child, C. C., III: Advances in Surgery, Vol. II, p. 495. Interscience Publishers, New York, 1949.
4. Pearse, H. E.: Surgery. **20**:663, 1946.
5. Owens, F. M., Jr.: Ann. Surg. **128**:15, 1948.

Discussion

Dr. William Ostrow (Brooklyn, N. Y.):—It is obvious that it would be ideal to make a diagnosis of carcinoma of the head of the pancreas before jaundice supervenes. This is a most difficult task.

Of course, I don't have to call the attention of an audience of this type to the necessity for the increased index of awareness of pancreatic malignancy. Everyone of us, whenever we come in contact with jaundice, accompanying any form of dyspepsia, thinks of the possibility of pancreatic malignancy. The impression that I have gathered over the years is that duodenal enzymes disappear much earlier than the serum enzymes begin to rise. As a matter of fact, the serum enzymes very rarely increase to very great levels; therefore, since the symptoms of malignancy of the head of the pancreas are not pathognomonic, and most of the time we become aware of it after jaundice ensues, I have thought that whenever we have a case of dyspepsia that cannot be explained on any other basis, we should begin to do duodenal enzyme studies. Whenever these are low, we should try to raise them by injections of secretin. If the secretin does not raise duodenal enzymes, then it behooves us to do serial and repeated blood enzyme studies.

If the serum enzymes begin to rise even in the absence of jaundice, I believe that we are justified in doing a laparotomy on these patients, obtain a biopsy, and

perhaps with this method, we might be able to pick up several cases per year of carcinoma of the head of the pancreas before obstructive jaundice appears.

Dr. Samuel L. Governale (Chicago, Ill.):—I should like to ask two questions:

1. Would the essayist comment on the incidence of biliary fistulae following the pancreatectomy; and

2. What is the incidence of wound dehiscence in the cases so presented?

Another question which I cannot resist asking is whether you drain the abdomen or not.

It seems obvious to us that a good number of the subtotal pancreatectomies or in the mobilization of the same in partial gastrectomy, dehiscence of the abdominal wound is increasingly encountered in our clinic. We are, indeed, at loss to explain this undesirable sequela; unless, however, it is due to the liberation of proteolytic enzymes from surgical trauma to the pancreas.

I should like to know what the essayist's experience is and suggest its preventative measures.

Dr. Stuart E. Krohn (Utica, N. Y.):—I should like to ask: How safe is it to take a biopsy of the pancreas, and how reliable are the pathological interpretations of these biopsies?

Dr. Sidney Winters (New Haven, Conn.):—I should like to ask the speaker one question: Upon what does he base his claim of operative procedure? I know that is a sort of an ambiguous question, but, nevertheless, I should like to know when should I suggest an operation, and what are the criteria which suggest nonoperative procedure, for individuals suffering with the condition under discussion.

Dr. Frank R. Fabiani (Dobbs Ferry, N. Y.):—I wish to emphasize a fact which I think some men may lose sight of in doing biliary surgery. If there is concomitant pathology of the common duct and the gallbladder, the pathology of the common duct should be first taken care of; in the event of a future procedure involving an anastomosis of the gallbladder to the gastrointestinal tract, the gallbladder is intact and can be used, whereas, if the cholecystectomy is performed first, and subsequently a carcinoma of the head of the pancreas is found, or a stricture, or some encumbrance to any further surgery, the gallbladder will already have been removed.

Dr. J. L. Chereskin (Springfield, Mass.):—In some of the cases where you do extensive resections, undoubtedly you get a development of diabetes. What do you do about it? How do you handle it, and how frequently does it occur?

Dr. Charles B. Ripstein (Brooklyn, N. Y.):—In answer to the first question, we have been rather lucky, I guess. We have sewed the patient up with through-and-through wire and kept the wire sutures in place for a long time, and we have used buried wire as well as wire stays. We haven't had any instance of wound dehiscence in this series.

As far as biliary fistula is concerned, we have employed drainage by suction through a stab wound in the flank, and placed a sump drain down to the area of the pancreas, and postoperatively we have kept them on suction drainage. The

general rule is, that the patient drains four or five days, and we can recover pancreatic fluid from the drainage. We have had very little biliary drainage, just in one case, and that was one where the common duct was double-ligated and not turned-in and inverted, as you would with a duodenal stump.

Since we have adopted that procedure, again with my fingers crossed, we have had no trouble, but drainage is a "must", as is the use of some nonabsorbable suture material which can be left in place for a long period of time.

Dr. Krohn asked a sixty-four dollar question. We have not found any difficulty with taking biopsies. I think that the great fear of biopsy in the pancreas is largely a bogeyman business. We haven't seen any harmful results occur from it.

So far as reliability of biopsies, it is unfortunately not 100 per cent. A positive biopsy is fine. There is no question of that as a rule, but I have recently had the experience of operating on a patient with a mass in the head of the pancreas which didn't quite feel like a carcinoma. The duct wasn't dilated too markedly, and two biopsies of that lesion showed chronic pancreatitis with no evidence of carcinoma, and a short-circuiting operation was done because I do not feel, in the absence of a positive diagnosis, it is justifiable to do such a radical procedure. The patient died nine months later from cancer of the pancreas, with metastases, so that one was wrong.

Frozen sections of the pancreas do not carry 100 per cent correlation with the paraffin sections, on autopsy or operative findings, so that although I don't think it is dangerous to biopsy the pancreas, it is very difficult to interpret the results, and certainly a negative biopsy always leaves a question of doubt.

As for Dr. Winters' question, the only thing I can say about when to advocate operation, is when you have any suspicion that the patient might have a carcinoma, whether that be on the basis of an obstructive jaundice which can't be explained in any other way, or vague dyspepsia, anorexia, with no duodenal enzymes.

As to deciding when to carry out the radical procedure at the operating table, that is a tough one. I really can't be 100 per cent sure, and, as I have recently found out, even the biopsies don't help sometimes, but, as a rule, the diagnosis can be made clinically by the feel of the lesion, by the appearance of it, by the presence or absence of gallbladder disease, by the state of the rest of the pancreas, and by the state of the common bile duct and the pancreatic duct, and I think that perhaps a clinical diagnosis is more accurate than frozen section, but it is not 100 per cent certain. I think we will all take out chronic inflammatory, and all leave in neoplastic lesions.

The point about the common duct is well taken. I have seen a case recently that had a cholecystectomy performed and, in the convalescence, when the jaundice didn't clear up, for, presumably, a stone in the common duct, a carcinoma was found which had been missed at the first operation.

In answer to Dr. Chereskin, in these resections of the head we have not seen diabetes. It does occur, in total pancreatectomy, but it is rather mild, and the

difficulty we have found is that it is so difficult to know what the insulin requirements are going to be from day to day. They change very rapidly and the patient who requires a large dose of insulin at 6:00 a.m., may be in hypoglycemia at twelve o'clock.

Once the diabetic state has stabilized itself, these patients require only forty to sixty of protamine zinc insulin lately, and the Mayo Clinic has reported two cases in which a diabetic has had pancreatectomy, and their diabetes was milder than it was before the removal of the pancreas.

Dr. Winters:—Were those two cases at Mayo, did you say, sir?

Dr. Ripstein:—Yes.

Dr. Fabiani:—Dr. Brunschwig reported several of those five years ago following radical surgery of the pancreas, that the diabetic state improved, and the explanation was not forthcoming.

RESULTS OF ROUTINE SIGMOIDOSCOPY*†

VINCENT T. YOUNG, M.D.

Washington, D. C.

The lower bowel is ignored in the process of routine physical examination more frequently than any other easily accessible region of the body. No logical explanation for this neglect can be found. There is irrefutable evidence that satisfactory examination of the lower bowel is of the utmost importance in every complete physical examination and should not be excluded. Malignant disease of the bowel furnishes a dramatic example of this point.

It has been estimated that 500,000 people currently suffer from cancer in this country[1]. Approximately 12 per cent, or 60,000, of these lesions are located in the anus, rectum, and sigmoid colon[2] which produce an annual mortality of 35,000[3]. Gass[4] has concluded that 70 per cent of these deaths could be prevented if proper investigation of the lower bowel was carried out as part of every routine physical examination. In other words, possibly 24,500 people die yearly in the United States because the lower bowel is neglected. Many physicians and surgeons will see fit to perform a digital examination of the rectum only if lower bowel symptoms are evident. But, when we must consider that in most instances early cancer is asymptomatic and that the well-known symptoms of bleeding, change in bowel habit, pain, loss of weight, and the usual constitutional alterations are late manifestations, this stand does not appear justified. It is well established that approximately 5 per cent of all people who are subjected to sigmoidoscopic examination will be found to have one or more polyps of the lower bowel[5]. Dukes[6] believes that all polyps eventually will become malignant. It is the opinion of Fitzgibbon[7] that all cancers of the bowel arise from polyps. Although Helwig[8] has presented evidence which would indicate that this may not always be so, most observers agree that the role of the polyp is paramount in the formation of cancer of the large bowel.

When a benign polyp of the lower bowel is detected by sigmoidoscopic examination it can usually be destroyed completely and permanently as a safe, simple office procedure which consumes but a few minutes and causes the patient little discomfort or inconvenience. An adequate follow-up should insure the patient against the danger of recurrence. Contrast this situation with that where cancer of the bowel is found. At best, the patient will be exposed to a formidable surgical procedure with only about a 25 per cent chance of a 10-year survival[9]. Therefore, the present answer to the problem of cancer of the lower bowel is the detection and treatment of the early precancerous lesion. This may be accomplished only by routine examination of the lower bowel.

*Read before the Fifteenth Annual Convention of the National Gastroenterological Association, New York, N. Y., 9, 10, 11 October 1950.

†From the Yater Clinic, Washington, D. C.

About 80 per cent of all intestinal cancers are within range of the sigmoidoscope[10] and 60 per cent are within range of the finger[11]. The same is essentially true of polyps[12]. However, most small polyps are not palpable, and a digital examination will not insure against the presence of these growths within the range of the finger by any means. The sigmoidoscope, on the other hand, adequately visualizes the mucosa of the bowel, and even small polyps should not be missed by the practiced operator. Therefore, digital exploration alone is not adequate. Satisfactory examination of the lower bowel must make use of the sigmoidoscope.

Unfortunately, most practicing doctors do not own a sigmoidoscope. Many of those possessing such an instrument seldom have occasion to use it. In fact, as a result of careful interrogation of patients, we must conclude that, in most instances, even the digital examination of the rectum is omitted from the general physical examination. Proctologists and gastroenterologists have long been aware of the importance of these facts and no competent individual in these specialties under any circumstance, neglects to sigmoidoscope every patient who comes under his care. As a rule, patients come to the proctologist only if they are suspected of having lower bowel disease, and not to have a lower bowel examination performed as part of a routine physical. Therefore, unfortunately, the proctologist ordinarily examines only a relatively small proportion of the total population.

In a clinic where a proctologic service is maintained undoubtedly a greater percentage of patients are subjected to adequate lower bowel examination than in a private practice. So far as can be ascertained at the present time, most clinics do not perform a sigmoidoscopic examination on every patient who does not present bowel symptoms. Even if we go on the assumption that every proctologist would consider this essential, it is understandable why this end is difficult to achieve. In spite of the fact that there is ample evidence to prove the point, it is hard to convince others of its importance. If every patient coming to a clinic was sigmoidoscoped, it would be necessary to expand the department in many instances to an extent that might seem unwarranted to the other services.

When the Yater Clinic was established in Washington, D. C. four years ago, a determined effort was made to establish the sigmoidoscopic examination as part of the routine physical. Essentially the same points which have been enumerated here were presented to the group in a plea for the adoption of this principle. The members of the group saw merit in the idea and agreed to adopt it on a trial basis. Its merit was soon proven and it has been enthusiastically accepted on a permanent basis.

This paper presents one year's experience with the routine sigmoidoscopic examination at the Yater Clinic, which, we feel, constitutes a strong argument for the universal adoption of this principle. In compiling the following data, more than 8,000 records were carefully examined and up to 500 of those patients who did not present any indication for examination of the lower bowel were selected

for study. All of these patients were first subjected to what was considered a thorough history and physical examination by the department of internal medicine. The usual questions regarding the lower bowel were included. In every instance in which the patient had a complaint which might be referable to the lower bowel, no matter how trivial, the case was excluded. For instance, many of these patients complained of minor degrees of constipation and other minimal symptoms which ordinarily would not have occasioned consultation with a proctologist. These were eliminated from this series. A digital examination was performed by the internist on every patient as part of his examination. If anything of a suspicious nature was discovered by this means, the case was not included.

When the patient reported for sigmoidoscopy a careful history referable to the lower bowel was again taken; this included considerably more detail than the usual medical history. If this interrogation revealed bowel symptoms which

TABLE I

Routine Sigmoidoscopy	Number	% of Total	Number of Females	Number of Males
Asymptomatic Patients Examined	500	100	318	182
Patients Showing Lower Bowel Lesions	138	27.6	78	60

were not recorded by the internist, the patient was included because it was felt that proctologic consultation would not have been specifically recommended in this instance. Of the 500 patients who satisfied these conditions 138 or 27.6 per cent were found to have lesions of the lower bowel (Table I).

Slight deviations from the normal, such as small hemorrhoids, deepened crypts, moderately hypertrophic papillae, etc., were not included in the tabulation. In many instances it was difficult to evaluate disease, and some interesting points were brought up in this connection. It must be remembered that this group was supposedly asymptomatic, and therefore should present few abnormal findings. However, the vagaries of human nature must be considered. Many of these patients undoubtedly had symptoms which they would not admit. Natural reticence in discussing matters pertaining to the "back passage" and fear of surgery are probably motivating factors in most of these instances.

The patients regarded as having lower bowel disease fell into the diagnostic groups indicated in Table II.

TABLE II

Diagnosis	Number of Cases	% of Cases Showing Lower Bowel Disease	% of Total Number of Cases Examined
Hemorrhoids	64	46.4	12.8
Polyps	44	31.9	8.8
Diverticula	9	6.5	1.8
Fissure	8	5.8	1.6
Hypertrophic Papillae	7	5.1	1.4
Pruritus Ani	7	5.1	1.4
Cancer of the Lower Bowel	5	3.6	1.0
Fistula	4	2.9	.8
Rectal Prolapse	4	2.9	.8
Melanosis Coli	3	2.2	.6
Condylomata Acuminata	2	1.4	.4
Nonspecific Proctocolitis	2	1.4	.4
Squamous Cell Metaplasia	1	.7	.2
Stricture	1	.7	.2
Rectocele	1	.7	.2
Radiation Proctitis	1	.7	.2
Blumer's Shelf	1	.7	.2

Hemorrhoids:—A large proportion of the adult population is found to have hemorrhoids, which, for the most part, are asymptomatic. Therefore, one does not consider small internal or external hemorrhoids particularly abnormal. Only large hemorrhoids which in my estimation would produce symptoms under ordinary circumstances were included.

TABLE III

Hemorrhoids	Internal Hemorrhoids	External Hemorrhoids	Internal and External Hemorrhoids	Totals
Number of Cases	6	3	55	64
%	9.4	4.7	85.9	100

Diverticula:—It has been well established that the presence of diverticula may be detected by sigmoidoscopic examination[13]. This was demonstrated in the

series by the finding of evidence of diverticula in 9 patients, in whom the diagnosis was later confirmed by x-ray examination.

TABLE IV

Diverticula of the Large Bowel	Age	Sex	Sigmoidoscopic Findings		Diagnosis	Symptoms
			Visible Diverticulum	Narrowing Fixation Thickening Loss of Elasticity (Lower Sigmoid)		
Case 1	74	F	No	Yes	Diverticulitis Colon	Bladder Irritation
Case 2	68	M	Yes	Yes	Diverticulitis Colon	Vague Abdominal Discomfort
Case 3	82	F	Yes	No	Diverticulosis Rectum	None
Case 4	44	M	No	Yes	Diverticulitis Colon	Vague Abdominal Discomfort
Case 5	28	M	Yes	No	Diverticulosis Colon	None
Case 6	54	F	Yes	No	Diverticulosis Colon	None
Case 7	36	F	No	Yes	Diverticulitis Colon	Vague Abdominal Discomfort
Case 8	38	M	No	Yes	Diverticulitis Colon	Two Episodes of Abdominal Cramps
Case 9	44	M	Yes	No	Diverticulosis Colon	None

One case was that of a 74-year-old woman who had suffered from bladder irritation for 10 years. She had been under the care of a competent urologist, and repeated studies had failed to reveal the cause of her symptoms. Marked improvement followed medical treatment of the diverticulitis. In another case a large solitary diverticulum was found bulging into the pouch of Douglas from the anterior wall of the rectum 11 cm. from the anus. A probe could be passed through the opening into the diverticulum. No treatment was deemed necessary.

It is reasonable to assume that these cases would have remained undiagnosed if routine sigmoidoscopy had not been performed.

Fissure:—Anal fissure was discovered in 8 instances. All of these showed deep ulceration with hard, indurated, fibrotic margins and indolent bases with excavation of the edges. In all cases bleeding could readily be produced by light swabbing. However, all of these patients steadfastly denied symptoms, a fact that was extremely difficult to understand. Surgery was urged in all these cases, but all of the patients refused it.

Hypertrophic Papillae:—Seven patients were found to have anal papillae which had hypertrophied to the point where prolapse through the anus was evident on straining. Excision was recommended in all cases and carried out in one.

Pruritus Ani:—Characteristic skin changes of pruritus ani were noted in 7 patients. On subsequent questioning, 5 of these admitted that they had suffered with various degrees of itching. Conservative treatment was recommended.

Fistula:—Four cases of uncomplicated anorectal fistula were encountered. No history of previous abscess or symptoms could be elicited. Operation was urged in each instance and accepted by 1 patient.

Rectal Prolapse:—This condition was seen in 4 patients. All expressed considerable surprise when informed of it. Injection treatment was urged in all of these cases. It was performed on 3 patients with excellent results.

Melanosis Coli:—The typical picture presented by this condition was observed in 3 instances. Subsequently the patients admitted taking cascara on frequent occasions.

Condylomata Acuminata:—Large accumulations of condylomata acuminata were seen in 2 cases. Both patients insisted that they were unaware that an abnormal condition existed and steadfastly denied all symptoms. Surgical excision was recommended and accepted by both patients.

Nonspecific Proctocolitis:—These 2 patients exhibited essentially the same picture, namely, slight injection and edema of the mucosa with infrequent, small, clean, shallow ulcers in the rectum and sigmoid. Extensive laboratory examination failed to establish a specific etiological factor. In both cases the bowel returned to normal within a month without treatment.

Stricture and Squamous Cell Metaplasia:—A stricture was discovered in a 61-year-old woman. This was located just above the pectinate line and would not admit a proctoscope of ½-inch diameter. The under surface showed raised, white areas on the mucous membrane which were found on microscopic examination to be squamous cell metaplasia of the rectum.

Radiation Proctitis:—A 61-year-old woman who had been exposed to irradiation of the uterus 11 years previously showed radiation proctitis.

Rectocele:—A large rectocele was discovered in an elderly woman.

Blumer's Shelf:—A 44-year-old man was found to have a well-defined Blumer's shelf. This patient was later found to have carcinoma of the stomach with metastasis to the rectovesical pouch.

Polyps:—It is well established that the polyp of the large bowel is a precancerous lesion. Therefore, it would appear that the present answer to the problem of cancer of the bowel is the discovery and treatment of the premalignant polyp. This can only be accomplished by subjecting the asymptomatic patient to adequate examination of the lower bowel. The following data on polyps accumulated in this study substantiate this (Table V).

TABLE V

No. of Cases Examined	No. of Patients With Polyps of Lower Bowel	%
500	44	8.8

Forty-four patients or 8.8 per cent of this asymptomatic group were discovered to have one or more polyps of the large bowel. None of the polyps was detected by digital examination. The location of the polyps is shown in Table VI.

TABLE VI

	Rectum		Rectosigmoid	Sigmoid
Inches From Anus	1 – 3	3 – 6	6 – 8	8 – 14
Anterior Wall	4	10	5	2
Posterior Wall	1	14	3	1
Lateral Walls	1	9	3	2
Total	39		11	5
Per Cent	70.9		20.0	9.1

The size distribution of the polyps is shown in Table VII.

TABLE VII

Size of Polyps	1 mm. to 5 mm.	5 mm. to 1 cm.	1 cm. to 2 cm.	2 cm. to 3 cm.	3 cm. to 4 cm.	Over 4 cm.
Number	31	14	7	1	1	1
%	56.4	25.4	12.7	1.8	1.8	1.8

Multiple polyps occurred as indicated in Table VIII.

TABLE VIII

Number of Polyps	1	2	3	4
Number of Patients	36	6	1	1
%	81.8	13.6	2.3	2.3

All small polyps were treated by fulguration without preliminary biopsy. Patients return for re-examination in two months and thereafter yearly. Barium enemas are ordered in all cases in which polyps are found. Microscopic examination is carried out on all polyps measuring more than 0.5 cm., regardless of gross appearance.

Cancer:—Cancer of the lower bowel was found in 5 patients or 1 per cent. This is quite surprising when one considers the fact that we are dealing with a strictly asymptomatic group.

TABLE IX

Cancer of the Lower Bowel	Age	Sex	Cm. From Anus	Gross Appearance	Microscopic Diagnosis	Treatment	Remarks
Case 1	82	M	20	Large, flat Cauliflower	Grade II Adeno-Carcinoma	Refused	None
Case 2	57	M	20	Large Annular	Grade III Adeno-Carcinoma	Miles Abdomino-Perineal Resection	Well 1 yr.
Case 3	33	F	13	Large, flat Papillary	Grade I Adeno-Carcinoma	Segmental Resection and Anastomosis	Well 9 mo.
Case 4	40	M	7.5	Small Polypoid	Grade II Adeno-Carcinoma	Local Excision	Well 1½ yrs.
Case 5	60	M	18	Large Annular	Grade II Adeno-Carcinoma	Miles Abdomino-Perineal Resection	Recurrence 1½ yrs.

One case was that of a 40-year-old doctor on whom a polypoid growth was found 7.5 cm. from the anus on the anterior wall of the rectum. This measured 1.5 cm. in diameter and was attached to the bowel by a fairly broad pedicle measuring 0.5 cm. It was soft, freely movable, and presented nothing remarkable in its appearance. Grossly, it appeared benign. The growth was snared off so close to

the base that a barely discernible elevation marked its location. Microscopic section showed adenocarcinoma, grade II, extending throughout most of the specimen but shading off to grade I at the base. An elliptical excision of the growth and the surrounding bowel wall was done. Over 300 serial sections through this specimen were carefully examined and no malignant change was observed. It is assumed that a small intervening section of the growth between the snared specimen and the bowel sloughed as a result of the snaring and that the malignant process had not yet advanced beyond this point. No further procedure was done. The patient has been followed carefully, and at this point 19 months following the surgery, there is no evidence of recurrence. It is logical to suppose that the bowel wall would have been invaded shortly and a radical resection of the bowel would have been necessary to save the patient's life.

At the present time all of these patients are alive, but one has recurrence. Because the survival time is short in the others, no definite conclusions can be reached. It cannot be denied that this group presents a much more favorable prognosis than if the lesions had not been discovered until after the onset of symptoms.

Summary

A significant number of asymptomatic patients are found to have cancer, precancerous growths, and other lesions of the lower bowel, on routine sigmoidoscopic examination.

REFERENCES

1. Bacon, H. E.: Anus, Rectum and Sigmoid Colon. J. B. Lippincott, Philadelphia, 3rd Edition, 1949, p. 600.
2. Smith, D.: Cancer of Anus, Rectum and Rectosigmoid. In: Cyclopedia of Medicine, Surgery and Specialities, Vol. 10. Philadelphia, Dave, 1934, p. 1106.
3. Lynch, J. M.: Rationale of the Surgical Technic in Carcinoma of the Rectum and Sigmoid. Am. J. Surg. **40**:4321, 1938.
4. Gass, O. C.: Precancerous Lesions of the Rectum and Colon: Diagnosis and Treatment. J. Tenn. State M. A. **39**:234, 1946.
5. Brust, J. C. M.: The Treatment of Polyps Above the Peritoneal Reflection. Tr. Am. Proct. Soc. **42**:213, 1941.
6. Dukes, C. E.: Simple Tumors of the Large Intestine and Their Relation to Cancer. Brit. J. Surg. **13**:720, 1926.
7. Fitzgibbon, G. and Rankin, F. W.: Polyps of the Large Intestine. Surg., Gynec. & Obst. **52**:1136, 1931.
8. Helwig, F. C.: Role of the Pathologist in the Diagnosis of Polypoid Lesions of the Colon and Rectum. Am. J. Surg. **76**:728, 1948.
9. Rankin, F. W.: The Curability of Cancer of the Colon, Rectosigmoid and Rectum. J.A.M.A. **101**:491, 1933.
10. Bacon, H. E.: Anus, Rectum and Sigmoid Colon. J. B. Lippincott, Philadelphia, 3rd Edition, 1949, p. 602.
11. Gabriel, W. B.: The Principles and Practice of Rectal Surgery. Charles Thomas, Springfield, Ill., 3rd Edition, 1946, p. 296.
12. Martin, W. J., Jr.: Polypoid Versus Carcinomatous Lesions of the Colon and Rectum. Southern M. J. **33**:428, 1940.
13. Buie, L. A. and Jackman, R. J.: Diverticula of the Colon. J.A.M.A. **121**:1144, 1945.

Discussion

Dr. Emil Granet (New York, N. Y.):—I am sure you will agree with me that Dr. Young has analyzed and presented his material in so excellent a manner that its great value to the gastroenterologist, the internist, and the surgeon, is

clearly evident. Just such tedious compilations of clinical material and personal experiences enable us to make great strides in the eradication of pathological lesions such as the precancerous rectal polyps which Dr. Young so ably discussed in his introduction.

Medicine in this era is dedicated to the prevention of cancer death by early detection of the lesion and its subsequent eradication. Members of our Association have a most prominent role in this cause. More than half of all cancers arise in the gastrointestinal tract. Recent statistics indicate that cancer of the intestine and rectum are being diagnosed more frequently than cancer of the stomach. As pointed out this morning by Dr. Young, 80 per cent of the colon cancers occur within the reach of the sigmoidoscope. It behooves all of us to make use of this valuable endoscope in all complete physical examinations, a primary aim of which should be cancer detection.

One other point. Rectal and colon cancers are not geriatric diseases. They occur commonly below the age of forty as separately reported by Buie, by Rafsky and by Rosser and Kerr.

To find a comparable group of rectally asymptomatic patients is not easy. It was fortunate that there were available at our office the records of patients referred to us for what we call a "gastrointestinal survey". This comprises roentgenography of the upper gastrointestinal tract, barium enema, gallbladder examination with dye, an adequate test meal, and finally sigmoidoscopy. Patients referred specifically for investigation of colon and rectal diseases were excluded, as were patients with specific anorectal complaints. Four hundred forty-seven patients were available who completed this survey, the majority of whom were in the middle age group.

This audience should be interested in the incidence of a few organic lesions found in these private patients. As you see, polyps were found on sigmoidoscopy in 21 or 4.7 per cent of our 447 patients. Of these 16 were sessile and 5 were pedunculated. Discrepancies between my numerical findings and those of Dr. Young probably are dependent on variations in age groups. Polyps, as do diverticula, occur more frequently in the older decades. Normally the anal papilla is barely visible. Hypertrophy of the papilla indicates past or present infection in adjacent crypts. All palpable and easily seen papillae were therefore reported, particularly to draw the referring doctor's attention to a probable anal focus of infection.

As we all know, and as Dr. Young pointed out, hemorrhoids are almost universally present in adults. Because of extreme variation in size, type, and associated lesions, accurate statistical evaluation is difficult. Consequently, listing of hemorrhoids was omitted in my survey.

Dr. Young was fortunate in finding diverticula by sigmoidoscopy in his nine patients. One might disagree with his statement, "It has been well established that the presence of diverticula may be detected by sigmoidoscopic examination". In my fifty-three patients with sigmoidal diverticula shown roentgenographically, I could not subsequently establish the presence of any of these through the sigmoidoscope. I have never seen either the sigmoidal opening of a diverticulum

or the sacculations of the mucosa described by Buie. Furthermore, Buie claims that angulation of the bowel which prevents passage of the sigmoidoscope is suggestive of diverticula. True it is that seven of our fifty-three patients had angulation which precluded examination higher than the distal sigmoid. Moreover, I found that either acute anterior angulation or fixation of the sigmoid mesentery limited the safe advance of the scope beyond the distal sigmoid in thirty-nine or 8.5 per cent of our 447 patients. In such cases, although visual examination is necessarily limited, I believe that above the rectosigmoid a competent roentgenographic examination by barium enema, with air contrast films, accurately will reveal significant lesions in the upper sigmoid and colon.

One patient seen recently, hospitalized with acute diverticulitis presented a constricted, edematous and inflamed granular mucosa at the rectosigmoid which indicated to me an inflammatory sigmoiditis, possibly diverticulitis. Two of my colleagues separately observed pus exuding from between folds of mucosa in patients with acute diverticulitis but neither observer actually saw the diverticular opening. Incidentally, Weber, of the Mayo Clinic, reports an incidence of 6 per cent for diverticulosis diagnosed roentgenographically. The incidence for this condition was 14 per cent in our cases.

Symptoms including constipation, low back pain, tenesmus, proctalgia following defecation and occasionally bloody mucoid discharge are commonly due to prolapse of the distal sigmoid into the rectal ampulla, a condition termed sigmoidal procidentia or sigmoidorectal intussusception. Although described repeatedly in proctologic text books since 1880, this condition is not commonly diagnosed. This is so because we ordinarily proctoscope patients in the "knee-chest" or equivalent positions in which "upside-down" positions the intussusceptum spontaneously reduces itself. When such patients are sigmoidoscoped in the left Sims' position, the sigmoid can be seen to invaginate into the rectal ampulla if the patient is asked to "bear down" while the scope is slowly withdrawn. It would be valuable to hear Dr. Young's experience with sigmoidal intussusception.

Not uncommonly seen on sigmoidoscopy, in the presence of cryptitis or other local inflammatory conditions of the sphincteric rectum, is an associated generalized hyperplasia of the submucosal lymph follicles of the ampullary rectum. The mucosa appears as if studded with "millet seed"-sized lesions, these definitely due to hypertrophy of the submucosal lymph follicles. I should like to hear Dr. Young's experience with this bizarre condition.

I express my gratitude for this opportunity to discuss Dr. Young's laudatory contribution.

Dr. Horace W. Soper (St. Louis, Mo.):—My slogan for the last forty years has been that no examination of a patient is complete without proctosigmoidoscopy.

I always use a Mayo flat table with the patient in the correct knee-chest posture, and Tuttle's modification of Strauss' instrument, with a light at the end, so that one can very easily do the diathermy for the polyps through this tube. I have my tubes measure all the way from three-quarters of an inch in diameter to three-eighths of an inch. Why do I have three-eighths? Because of strictures of

the rectum, and so forth. You sometimes can't get the larger instruments through; furthermore, every time we do a sigmoidoscopy, we do a vaginoscopy, and in the virgin you can, regardless of the hymen, pass your three-eighths tube. Very frequently the cervix of the uterus has polyps which you can easily destroy with diathermy.

Never use radium in any of these lesions, as the diathermy spark is so much better controlled and leaves no contracture.

Another thing to remember is that the anal canal is one of the most sensitive regions of the body and many patients complain of the severe pain they experienced in former sigmoidoscopies.

We have found that a medicine dropper, filled half full with 10 per cent solution of cocaine, introduced in the anal canal and allowed to remain in there ten minutes before your examination, is helpful. There is absolutely no pain experienced by the patient in passage of the instrument. That is a very valuable thing for anyone who is going to do a proctosigmoidoscopy. I never see any bad after-effects from it, and it is very effective.

Dr. Vincent T. Young (Washington, D. C.):—I should like to compliment Dr. Granet on his very excellent and comprehensive discussion. He asked me to make a few remarks about sigmoidal prolapse. Unfortunately, I have had very little experience with it. I am forced to admit that I have never yet encountered a severe type as pictured by Dr. Granet. Now, in this series I have probably encountered about ten cases where, let's say, there was slight prolapse, but I did not call it that; I simply called it redundancy, and I did not consider it particularly abnormal. So far as I know, no symptoms were produced by the condition, so I did not record it as a pathologic entity.

In closing I should like to make a plea. It is that when we all leave this meeting and return to our respective communities we be just a bit more persistent than we have been in preaching the gospel to the surgeons, internists and practitioners, of the importance of the sigmoidoscopic examination as part of the routine physical examination.

HABITUAL PYLORIC SPASM

Case Report

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"Habitual pyloric spasm" may simulate carcinoma of the stomach. Tracey reports eleven such cases, seven of which had exploratory laparotomy, because the problem could not be safely decided without this procedure.

This case report adds another to the above series. Our patient, M. C., age 63, was referred by Dr. Louis Pellman for study because of intractable epigastric pain. The pain was postprandial and relieved by intake of bland foods. There was a loss of fifty pounds in six months time but this could have been the result of low caloric intake with intent to reduce overweight. Barium study of the

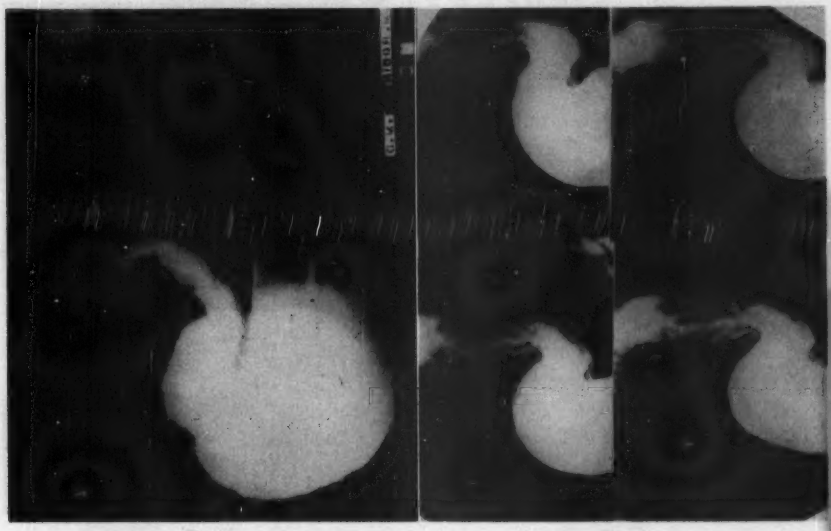

Fig. 1 Fig. 2

Fig. 1—Lateral view, Preoperative Film, April 1948.
Fig. 2—M. C., Preoperative, April 1949.

stomach by fluoroscopy and x-ray film revealed no abnormality up to the antrum. The pars pylorica appeared to be rigid permitting no peristaltic waves to pass through. The appearance of this area was somewhat like the profile of a duck, the greater curvature forming the breast, neck and beak. The rugae appeared to be intact in this area and retained their mucosal pattern.

The above description answered the criteria of infiltrating lesion particularly when several such examinations over a period of seven months revealed no increase in distensibility or change in contour or mucosal pattern. Clinically, the picture was one of a persistent pathology since the pain continued during the entire period of observation.

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Gastroscopy, performed by the writer, revealed a narrowing of the distal portion of the stomach. The rugae in the antrum could not be satisfactorily observed. The impression then was narrowing of the distal segment due to either localized gastritis or carcinoma.

Following the procedure described by Tracey, our patient was subjected to laparotomy by Dr. Harry Mackler. The operative findings were as follows: "The distal six centimeters of the stomach is somewhat tubular in appearance compared to the remainder of the stomach. The wall, however, is soft and not indurated in any part. No evidence of thickening or of ulcers as in gastritis. The stomach was opened and palpated by finger with negative findings. A posterior gastroenterostomy was done."

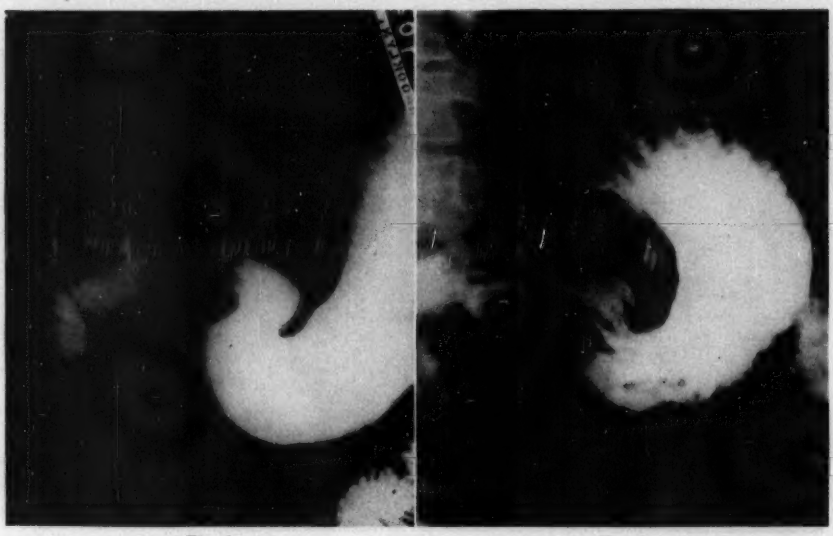

Fig. 3 Fig. 4

Fig. 3—M. C., Preoperative, Nov. 1949.
Fig. 4—M. C., Postoperative, Nov. 1949.

Patient felt well for several weeks postoperatively and then had another episode of epigastric postprandial distress. At this time the stomach was re-x-rayed and aside from the presence of a gastroenterostomy, revealed the same narrowed, fixed appearance of the antrum.

Films taken after atropine sulphate, grains 1/50, prostigmine bromide 1-2000, aminophyllin grains seven and a half, failed to reveal any change in the appearance of the antrum.

Conclusions:—Where there is a narrowing of the antrum that persists with little or no change over a period of months and through many roentgenologic examinations, the defect may simulate organic disease. Gastroscopy is of no value in the differential diagnosis. Laparotomy seems to be the only method of making a positive diagnosis.

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REF.:
1) Jones, C. M., et al.: Ann. Int. Med. 29:1-10, July 1948
2) Becker, G. H., et al.: Gastroenterology 14:80-91, Jan. 1950

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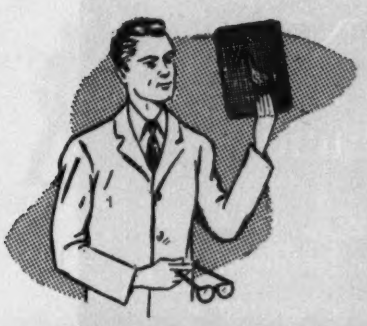

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"Dependability Through the Years"

relief!

the measure of a good hemorrhoidal suppository . . . . . .

ANUSOL* *HEMORRHOIDAL SUPPOSITORIES*

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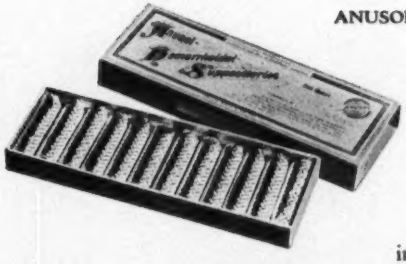
Preferred and Prescribed by Physicians for more than half a Century.

ANUSOL* HEMORRHOIDAL SUPPOSITORIES promptly and effectively relieve the pain and discomfort of the common anorectal disorders.

ANUSOL* HEMORRHOIDAL SUPPOSITORIES do not contain narcotic or analgesic drugs which may mask more serious anorectal disorders.

For best results one **ANUSOL*** in the morning and at bedtime and immediately following each evacuation.



ANUSOL* **HEMORRHOIDAL SUPPOSITORIES**, individually foil wrapped, are available in boxes of 6, 12 and 48.

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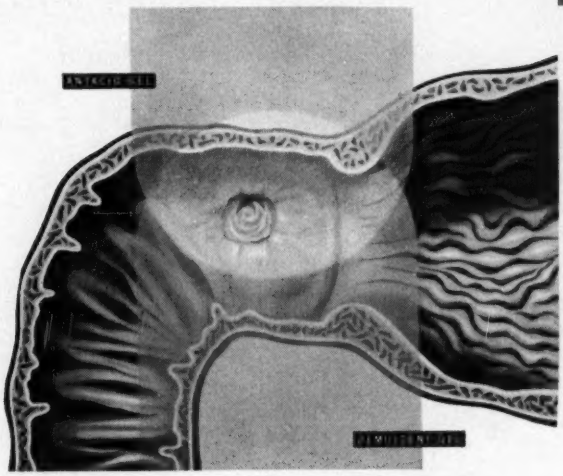

AMPHOJEL Has Many Important Advantages

for the successful medical management of acute or chronic peptic ulcer. These include:

1. Relieves pain in minutes
2. Promotes rapid healing of the ulcer crater
3. Does not interfere with digestion
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5. Does not inhibit the action of antispasmodics or laxatives if and when these are indicated
6. Safe to take for long periods of time
7. Pleasant and convenient to take
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